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APPENDICES
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COMMENTS ON THE METRIC SYSTEM

The metric system is used to describe weights and measures. It is a decimal system -- units are consistantly named to reflect multiplication or division of the basic unit by some power of 10 (10, 100, 1000, etc.). The two basic defined units of this system are the meter and the kilogram. All other units (volume, area, etc.) are calculated based on these two.

Metric measurement is the standard for scientific communication worldwide. It has been selected in preference to many other systems including the English system of measures which is in common use in the United States. Below is a table which will help readers of this Risk Assessment understand the metric numbers presented throughout the text.

ABBREVIATIONS
(Metric and English)

ac = acre
cc = cubic centimeter
cm = centimeter
ft = foot
g = gram
ha = hectare
in = inch

kg = kilogram
km = kilometer
l = liter
lb = pound
m = meter
mg = milligram
mi = mile

ml = milliliter
mm = millimeter
ppm = parts per million
oz = ounce
qt = quart
um = micrometer

CONVERSIONS

Length:

METRIC to ENGLISH
1 km (1,000 m) == 0.6214 mi
1 m == 39.37 in
1 cm (.01m) == 0.394 in
1 mm (.001m) == 0.0394 in
1 um (.000001m) == 0.000039 in

ENGLISH to METRIC
1 mi == 1.609 km
1 ft == 0.305 m
1 in == 2.54 cm

Mass / Weight:

1 kg (1,000 g) == 2.2046 lb
1 g == 0.035 oz
1 mg (.001 g) == 0.000035 oz
1 ug (.000001 g) = 0.000000035 oz

1 lb == 453.592 g
1 oz == 28.35 g

Others:

1 l == 1.056 qt (liquid)
1 ha == 2.471 ac
1 kg/ha == 0.89 lb/ac

1 qt == 1.136 l
1 ac == 0.40 ha
1 lb/ac == 1.12 kg/ha

APPENDIX A

Risk Assessment

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APPENDIX A

RISK ASSESSMENT FOR THE USE OF HERBICIDES IN THE SOUTHERN REGION, USDA FOREST SERVICE

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Section 1

INTRODUCTION

PURPOSE

The purpose of this risk assessment is to document probable effects on human health, wildlife, and aquatic species that could result from use of the herbicides 2,4-dichlorophenoxyacetic acid (2,4-D), 2-(2,4-dichlorophenoxy)propionic acid (2,4-DP), dicamba, fosamine, glyphosate, hexazinone, imazapyr, picloram, sulfometuron methyl, tebuthiuron, and triclopyr and the herbicide adjuvants kerosene, diesel oil, and limonene in vegetation management programs on National Forests and National Grasslands in the Southern United States (Forest Service Region 8). The risk assessment is developed as an appendix to environmental impact statements (EIS's) for vegetation management being prepared for three major southern physiographic areas: the Coastal Plain/Piedmont, the Appalachian Mountains, and the Ozark/Ouachita Mountains. The EIS's analyze the environmental impacts of using various alternatives for managing vegetation in Southern National Forests.

ORGANIZATION OF THIS APPENDIX

This document is organized as follows:

Section 1 presents the purpose, describes the structure, and outlines the methodology of the risk assessment.

Section 2 outlines vegetation management programs that use herbicides and application methods and mitigation measures practiced in each.

Section 3 summarizes and discusses the toxic properties of each herbicide for humans, including the cancer potency of the known or suspected carcinogenic herbicides.

Section 4 describes the methods used to estimate levels of human exposure and resultant acute and long-term doses to workers and the public.

Section 5 analyzes the human risk by comparing the results of the exposure analysis with the toxic effect levels described in section 3. Section 5 also discusses cancer risk, based on estimated lifetime doses to workers and the public, and the risks of heritable mutations, synergistic and cumulative effects, and the potential for effects on sensitive individuals.

Section 6 describes the herbicides' toxic effects on wildlife and aquatic species.

Section 7 discusses how wildlife and aquatic species' exposures were estimated.

Section 8 discusses the risk to wildlife and aquatic species in general and to sensitive species, particularly the red-cockaded woodpecker, gopher tortoise, and smoky madtom.

OVERVIEW OF THE HUMAN HEALTH RISK ASSESSMENT

The human health risk assessment consists of comparing doses that people may get from applying the herbicides (doses to workers) or from being near an application site (doses to the public) with doses that have produced no observed toxic effects in test animals in controlled laboratory studies. Risk judgments are based on the size of the ratio between the laboratory dose and the estimated human dose--called the margin of safety (MOS). In general, MOS's of 100 or greater indicate negligible risk to workers and the general public (EPA, 1986). The risk assessment analyzed the health effects of the active ingredient of each herbicide in various liquid, granular, or pellet formulations and the effects of light fuel oils (kerosene and diesel oil) and limonene. Kerosene is an inert ingredient in some formulations of 2,4-D and triclopyr; diesel oil is used as a carrier; limonene is used as an adjuvant.

Wilson and Crouch (1987) suggest that the task of the risk assessor is to use whatever information is available to determine whether an effect may result from some hazard, such as a chemical introduced into the environment, and to present a judgment ranging between the extremes of virtual certainty that the effect will not occur (risk = 0) to virtual certainty that the effect will occur (risk = 1). The human health risk assessment uses a conservative approach that tends to exaggerate estimated risks to human health. Assumptions about herbicide applications and about herbicide movement and degradation tend to overestimate doses that workers and the public would be likely to receive. Toxicity levels used to judge risks were dose levels where no systemic or reproductive effects were seen in the most sensitive laboratory test animals.

Wilson and Crouch (1987) state that:

> ...preventive public health suggests that we endeavor to estimate risks even where no historical data exist and the risk is small. This is often done by analogy with the cancer risks to animals, usually rodents, which are deliberately exposed to large enough quantities of pollutant so that an effect is observed. To use these data to estimate the risk at low doses in people involves (to oversimplify matters) two difficult steps: the comparison of carcinogenic potency in an animal and man and the extrapolation from a high dose to a low dose.

Cancer potencies were derived from data on the species and sex with the highest tumor rate. In addition, the value derived from the model that used the potencies to quantify cancer risk was the upper 95-percent risk level. This conservatism, both in estimating exposures and in setting and extrapolating from toxicity levels, led to an overestimation of the real risks of the herbicide application program so that any errors made would be on the side of safety.

Structure of the Human Health Risk Assessment

The risk assessment methodology used in the analysis of the human risks of herbicide use in Region 8 is the one generally recognized by the scientific community (National Research Council (NRC), 1983) as necessary to characterize the potential adverse human health effects of hazards in the environment. This method employs three principal analytical elements--hazard analysis, exposure analysis, and risk analysis. Dose-response assessment (presented as a separate step in NRC, 1983) is a key part of the hazard analysis.

(1) **Hazard Analysis** requires gathering information about the toxic properties of each chemical. Human hazard levels are derived primarily from the results of laboratory experiments on animal models, such as rats, mice, and rabbits, supplemented where appropriate with information on human poisoning incidents, field studies of other organisms, and data on chemical structure. Dose-response assessment (presented as a separate step in NRC, 1983) is a key part of the hazard analysis.

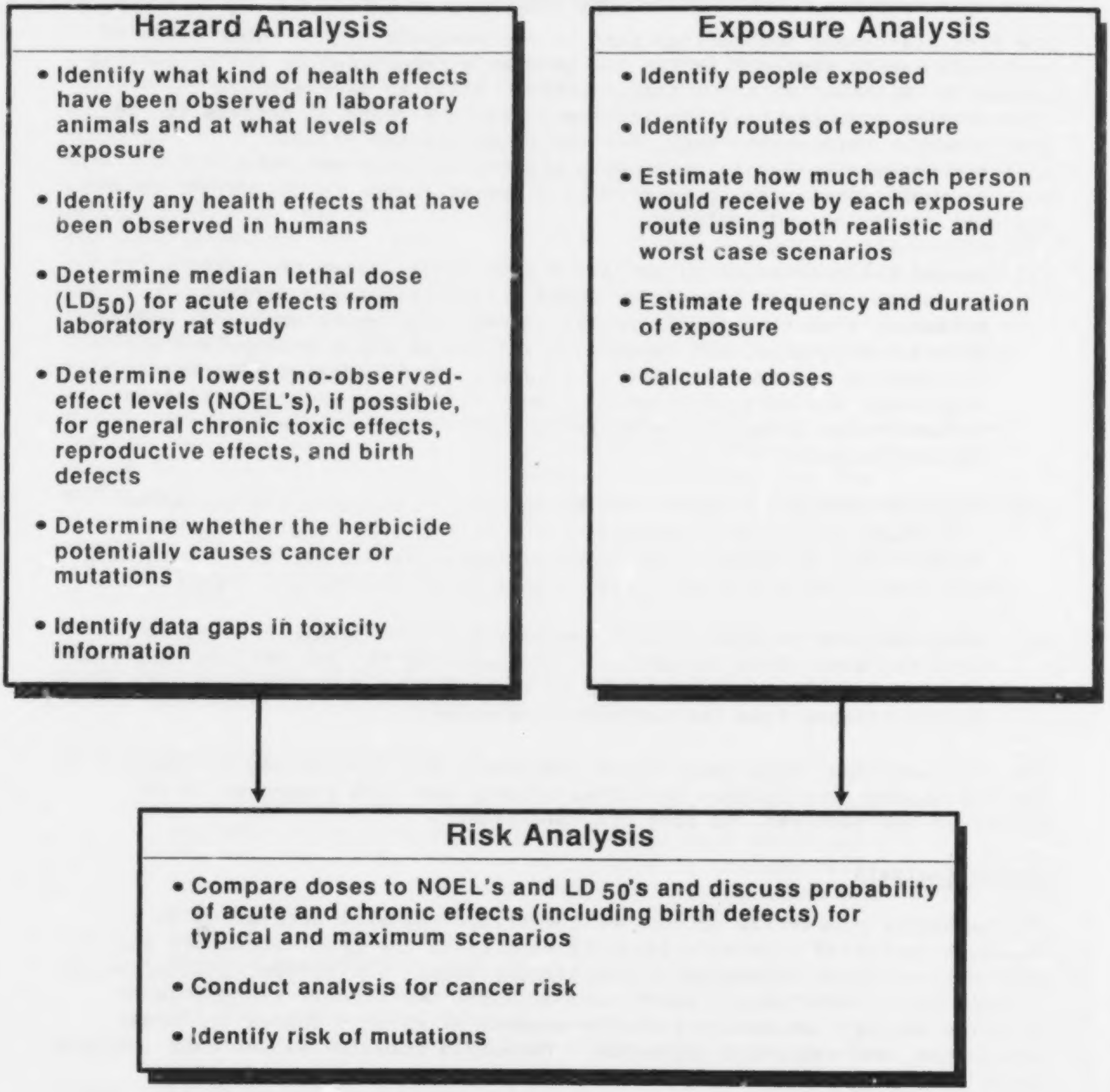
(2) **Exposure Analysis** involves estimating single and multiple exposures to persons potentially exposed to the herbicides, determining the doses likely to result from those estimated exposures, and determining the number and characteristics of persons in the exposed populations.

(3) **Risk Analysis** requires both a comparison of the hazard information with the dose estimates and an examination of the probability that the exposures could occur to determine the likelihood and severity of health effects from the estimated exposures.

The relationships among these three components are illustrated in figure 1-1. The discussion that follows describes briefly how each component in the structure was addressed in this risk assessment.

Hazard Analysis

The hazardous properties of each of the herbicides were determined in a thorough review of available toxicity studies in the open literature and of publicly available summaries of proprietary data. The review included acute (single dose), subchronic (short-term dosing), and chronic (long-term or lifetime dosing) laboratory toxicity studies of effects caused by dermal, inhalation, and ingestion exposures. Threshold toxicity values that included acute oral LD_{50}'s (median lethal dose) and systemic and reproductive no-observed-effect levels (NOEL's) were determined for each herbicide. The hazard analysis also reviewed available results of mutagenicity assays and cancer studies and developed cancer potency values for 4 of the 11 herbicides (2,4-D, 2,4-DP, glyphosate, and picloram) that had indications of potential carcinogenicity in animals. A cancer potency also was estimated for the light fuel oils, kerosene, and diesel oil, which contain small amounts of substances known or suspected of causing cancer. Scientific uncertainty



Hazard Analysis

- Identify what kind of health effects have been observed in laboratory animals and at what levels of exposure
- Identify any health effects that have been observed in humans
- Determine median lethal dose (LD_{50}) for acute effects from laboratory rat study
- Determine lowest no-observed-effect levels (NOEL's), if possible, for general chronic toxic effects, reproductive effects, and birth defects
- Determine whether the herbicide potentially causes cancer or mutations
- Identify data gaps in toxicity information

Exposure Analysis

- Identify people exposed
- Identify routes of exposure
- Estimate how much each person would receive by each exposure route using both realistic and worst case scenarios
- Estimate frequency and duration of exposure
- Calculate doses

Risk Analysis

- Compare doses to NOEL's and LD_{50}'s and discuss probability of acute and chronic effects (including birth defects) for typical and maximum scenarios
- Conduct analysis for cancer risk
- Identify risk of mutations

Figure 1-1--Components of the risk assessment process regarding the results of these studies (for example, concerning the results of the cancer studies on glyphosate and 2,4-D) also is discussed.

The review also identified toxicity information that is missing or unavailable for each herbicide. In such cases, judgments may be made about toxic properties based on other types of studies. Judgments about mutagenicity may be based on the results of cancer studies; those about carcinogenicity may be based on other chronic or subchronic studies. Conclusions also are drawn from chemical structure-activity relationships and from the known toxicity of similar chemicals. The hazard analysis is presented in section 3.

Toxicity information is summarized for 7 of the 11 herbicides in the background statements in Agriculture Handbook No. 633 (U.S. Department of Agriculture (USDA), 1984). Tebuthiuron toxicity is reviewed in the first supplement to Agriculture Handbook No. 633 (USDA, 1986). Toxicity information for the herbicides imazapyr and sulfometuron methyl and for the light fuel oils is summarized in background statements written in conjunction with this risk assessment. These documents are incorporated by reference into this report in accordance with 40 CFR 1502.21 and are available for review at all USDA Forest Service Offices in Region 8, as well as at the address shown on the cover page.

Exposure Analysis

Herbicide exposures and resultant doses to workers and the public in activities related to Forest Service Region 8 applications were estimated in the exposure analysis. Exposure scenarios (simplified descriptions of herbicide application operations and of potential routes of human exposure) were used to estimate a range of possible exposures (typical, maximum likely, and accident). Typical application scenarios were used to estimate the average doses to workers and to nearby members of the public that may reasonably be expected to occur during routine operations. Maximum scenarios were used to estimate the highest doses that are realistically expected to occur and that are not likely to be exceeded except in the case of an accident. Both typical and maximum doses then are considered realistic dose estimates. Accident scenarios were used to estimate doses to workers and the public that may result from direct exposure to herbicide spray mix or concentrate or from drinking water into which a helicopter load of herbicide mixture or a container of herbicide concentrate has been spilled.

Herbicide Application Operations

To estimate potential human exposures to the 14 herbicides and additives, major aspects of the vegetation management programs that determine herbicide exposure levels were identified, including types of formulations, application methods, application rates, target vegetation, size and configuration of spray areas, and mitigation measures. Descriptions of the methods are given in section 2.

The herbicides examined in the risk assessment can be applied aerially or on the ground using mechanized equipment or hand-held devices. Aerial applications use helicopters primarily for silviculture, right-of-way, and range management. Ground mechanical applications use truck- or tractor-mounted booms or other spraying devices for right-of-way and silviculture projects. Ground manual methods include basal applications using full-basal or streamline techniques; soil spot treatments using grid or root collar placement of herbicide; direct foliar applications; and cut-surface treatments using injection, frill or girdle, or cut-stump techniques. Herbicides also may be applied in solid formulation as granules or pellets, or as a liquid mixture carried in backpack canisters or in hand-held squeeze bottles. These methods are described in section 2.

To be conservative, the cumulative analysis assumes that 242,817 hectares (ha) (600,000 acres (ac)) are treated in the Forest Service's Southern Region each year in the vegetation management program. The size of the program and mix of activities may vary in any given year as described in the parent EIS. Table 1-1 gives a summary of the approximately 44,676 ha (110,350 ac) treated with herbicides in Region 8 during 1986 for various vegetation management programs. Individual silviculture treatment units within a project typically range from 10.1 to 24.3 ha (25 to 60 ac). Occasionally there are treatment areas much smaller (less than .4 ha or 1 ac) or much larger (up to 202.3 ha or 500 ac), especially on wildlife habitat rehabilitation projects. Treatment units for range management projects are somewhat larger than for silviculture, with 16.2 to 404.7 ha or more (40 to 1,000 or more ac) treated in a single project. Treatment areas for maintenance of facilities are typically very small, ranging from less than a square meter to a fraction of a hectare. The total area treated with various vegetation management treatments in 1986 was about 5 percent of the 4,856,333 ha (12,000,000 ac) of National Forest land and National Grasslands in Region 8. Further details about these operations are given in the body of the EIS's and in section 2.

Table 1-1

Acreage of herbicide spraying operations for
Region 8 lands in 1986

Purpose	Hectares	Acres
Conifer release	17,338	42,825
Hardwood release	1,620	4,000
Weed control (herbaceous, noxious, and poisonous)	860	2,125
Range improvement	486	1,200
Right-of-way maintenance	668	1,650
Site preparation	21,103	52,125
Precommercial thinning	1,549	3,825
Wildlife habitat improvement	1,052	2,600
Total	44,676	110,350

Affected Populations

The risk assessment examines potential health effects of herbicide use on two groups of people who might be exposed to the 11 herbicides and 3 additives in activities related to vegetation management programs: workers and the general public. Workers include personnel directly involved in herbicide applications: mixers and loaders, tractor or truck-spray or granule applicators and drivers, backpack sprayers, hand spray or granule applicators, pilots, observers, and supervisors. The public includes forest visitors and nearby residents who may inadvertently be directly exposed to herbicide as a result of drift or by being accidentally sprayed or indirectly exposed by contact with herbicide on plant surfaces or by eating food items or drinking water containing herbicide residues.

Exposure Scenarios

This risk assessment examines the health effects of exposure to an individual herbicide treatment, as well as the cumulative effects of exposure over a number of years. To represent the range of doses under normal operating procedures, typical and maximum application scenarios are used. In typical scenarios, application methods employing normal herbicide application rates and typical treatment unit sizes are used to calculate doses to workers. Doses to members of the public who may be in the area or who may live nearby are calculated for aerial or ground mechanical and broadcast methods. No direct public exposures are expected from granular or hand-application treatments because drift is negligible from these methods and no visitors are expected to be onsite during vegetation management activities.

Additional scenarios, using the same application methods as in the typical scenarios but employing the highest application rates likely to be used and the largest treatment unit sizes under conditions conducive to offsite herbicide drift, are used to estimate the maximum realistic doses to workers and the public. These estimates of exposure purposefully overestimate doses expected from routine applications.

Cumulative doses were estimated by using information on typical and maximum treatment days per year and on typical and maximum number of years exposed for workers and the public.

Accident Exposure Scenarios

Because all human activities involve the possibility of error, use of herbicides in vegetation management involves the possibility that humans may inadvertently receive unusually high exposures to the herbicides because of accidents. The types of accidental exposure analyzed in the risk assessment included direct aerial application of herbicide on a person, spills of herbicide concentrate on workers in mixing and loading, and spills of herbicide into drinking water supplies.

The likelihood that the events described in each accident scenario would actually occur was also examined. Wherever possible, historical records of accidents were used in determining the probabilities of accident occurrence.

Dose Estimation

Estimates of routine doses to workers were derived from field studies on the herbicide 2,4-D because it has been investigated under a variety of application conditions and its metabolism and dermal penetration are relatively well known. Suitable worker exposure data are not available for most of the 11 herbicides, so doses were extrapolated from a 2,4-D worker exposure study that used the same application method. Forestry worker exposures were extrapolated from the most similar studies of field operations because no exposure data exist for many of the ground methods used in Region 8.

Worker exposures to each herbicide were based on the worker's task (for example, backpack sprayer, pilot, mixer/loader) rather than the type of vegetation management project because the same equipment and procedures are used in these projects. The exposures between operation types are weighted by application rate and number of hours worked per day. Where the exposure of a worker in a particular task, such as a mixer/loader, is significantly different from one project type to another, that exposure is determined separately for each representative operation.

Exposures and doses to members of the general public were derived by using data on herbicide drift from field studies and by applying various assumptions about dermal penetration, amount of skin exposed, and diet. Details of the exposure analysis are given in section 4.

Risk Analysis

The risk analysis was conducted after the worker and public exposures were estimated by comparing the estimated typical, maximum, and accident scenario-based doses with the toxicity levels found in the hazard analysis. For threshold effects, the doses were compared to systemic and reproductive NOEL's determined in the most sensitive test animal species. A margin of safety, which is the animal NOEL divided by the estimated human dose, was computed to relate doses and effects seen in animals to estimated doses and possible effects in humans. For example, an animal NOEL of 20 milligrams per kilogram (mg/kg) divided by an estimated human dose of 0.2 mg/kg gives an MOS of 100. A margin of safety of 100 is comparable to the 100-fold safety factor that is the generally recognized value for setting safe doses for humans from valid long-term laboratory animal studies. The larger the margin of safety (the smaller the estimated human dose compared to the animal NOEL), the lower the potential risk to human health.

For the herbicides that could possibly cause cancer, a person's lifetime cancer risk was based on animal studies that related the chances of developing tumors to increasing herbicide doses. The analysis showed that currently there is scientific uncertainty regarding the potential of four of the herbicides--2,4-D, 2,4-DP, glyphosate, and picloram--and the light fuel oils to cause cancer in humans. The risk of cancer from a given lifetime level of exposure to any of these herbicides, is based on an estimated total lifetime exposure to the herbicide averaged to a daily exposure over a 70-year lifetime. The total lifetime exposure used in calculating the average daily dose could be to workers exposed over many years as applicators or to members of the public who may have only a single lifetime exposure. The average daily dose is multiplied by a cancer potency value derived for the herbicide in question from laboratory animal data on tumor incidence at increasing dose levels. These data are adjusted for species differences, body size difference, dose frequency, and duration of exposure.

Current scientific knowledge does not allow a quantification of mutagenic risk. Thus, the risks of heritable mutations are discussed qualitatively using available test data on bacteria, yeasts, plants, mammalian cells in culture, and whole animals. Where no test data are available, these herbicides are assumed to be mutagenic, and their risk of causing heritable mutations is compared to the herbicide's cancer risk.

Cumulative risk for individuals is discussed in terms of lifetime exposures for workers and for members of the public. Risk of synergistic effects is discussed in terms of available evidence of enhanced toxicity in mixtures of two or more herbicides. Risk to sensitive individuals is discussed qualitatively in terms of the likelihood of a sensitive individual being exposed.

A number of data gaps and areas of uncertainty were identified in the course of preparing this risk assessment. Field data on worker exposures to any of the herbicides are limited. No field data on public exposures are available. A number of specific types of toxicity studies are not available for several of the herbicides. In these instances, an extrapolation from existing data on a surrogate chemical had to be made, or a modeling of the herbicide's behavior was done. A dermal penetration rate of 10 percent is used, based on the known penetration rates of 2,4-D and picloram, for the herbicides for which no data are available.

Judging risks to human health from the Forest Service program involves several areas of uncertainty. First, the safe levels used in comparing estimated exposures are the results of toxicity tests on laboratory animals, particularly rats and mice, where dose levels produce no observed effects. To allow for the uncertainty in extrapolating from these no-observed-effect levels in laboratory animals to safe levels for the general population, additional safety factors are used. The generally accepted factors (NRC, 1986) are 10 for moving from animals to humans (between species variation) and another 10 to account for possible variation in human responses (within species variation). This 10 times 10, or 100-fold, safety factor means that the laboratory NOEL dose reduced 100-fold would normally be considered a safe dose to the general public, including most sensitive individuals; an additional safety factor of 10 (giving an MOS of 1,000) may be used to ensure that sensitive individuals are further protected. In this risk assessment, a margin of safety has been calculated for each estimated dose by dividing the animal NOEL by the estimated dose. The computed MOS is then compared to the 100-fold safety factor to judge risks of toxic effects.

A second area of uncertainty is in judging the risk to humans of doses that may be received once or perhaps a few times in a person's life (accidental worker doses and all doses to the public fall into this category). These risks were evaluated by comparing those human doses to levels of the herbicide that produced no ill effects in laboratory animals, even though the animals received the doses every day of their lives. This risk assessment is conservative because it uses the MOS approach discussed above in comparing one-time human doses to lifetime animal doses in all cases, even though this leads to an overestimation of the risks.

A different approach is used to assess the risks to humans from the herbicides or additives that may cause cancer. These chemicals are assumed to have some risk even at extremely low doses. In this case, a cancer potency value, expressing the probability of developing tumors at increasing dose levels, is taken from laboratory animal studies and adjusted for the differences in body weight and lifetime duration between the animals and humans. This potency, multiplied by an estimated human lifetime dose, provides an estimate of human cancer risk. The risk assessment uses the upper bound (95-percent level) of potency to quantify cancer risk.

A third area of uncertainty involves the estimation of human doses likely to occur in herbicide use. This risk assessment has been designed to overestimate doses and thus to err on the side of safety. In reality, workers are likely to receive lower doses than estimated. Standard safety practices and the use of protective clothing and immediate washing in the case of a spill normally will reduce actual dose levels below those estimated in this analysis. No member of the public is likely to receive as high a dose as estimated in this risk assessment again because typical safety practices and the remoteness of most treated areas limit the probability of any public exposure. Other assumptions made to ensure that doses are overestimated include assumptions that no herbicide degradation occurs, members of the public do not wash themselves or their food items after a spraying, and the public consumes water that has received herbicide from drift or a spill immediately after the event. Thus, the way in which exposures are estimated in this risk assessment and the way risks are judged both tend to exaggerate actual risks.

Wildlife and Aquatic Species Risk Assessment

The analysis of risks to wildlife and aquatic species was conducted in a manner similar to the human health risk assessment. The basis for comparison, as suggested by the U.S. Environmental Protection Agency (EPA, 1986) in their document on environmental risk assessment, is the species LD_{50} or LC_{50} (median lethal concentration). The Region 8 risk analysis uses laboratory toxicity data on species most closely related to a series of representative wildlife and aquatic species of the National Forests of the Southeast. Details of the analysis are presented in sections 6, 7, and 8.

Section 2

VEGETATION MANAGEMENT PROGRAMS

Region 8 encompasses the states of Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and a small area of West Virginia. This region has approximately 4,858,000 ha (12,000,000 ac) of National Forests and National Grassland. Vegetation management is conducted on approximately 3,600,000 ha (9,000,000 ac), with 243,000 ha (600,000 ac) treated annually.

This section describes the annual vegetation management programs involving the use of herbicides that the Forest Service conducts in Region 8 on approximately 44,535 ha (110,000 ac). Application methods, equipment, and herbicides used in those programs are identified. In addition, mitigation measures used to minimize the possible adverse effects of the herbicides on human health and the environment are described. Herbicide application rates for the different methods are given in section 4. Complete descriptions of the Forest Service vegetation management programs are in the environmental impact statements that this document supplements.

PROGRAM DESCRIPTIONS

The Forest Service conducts vegetation management programs to sustain and improve the ability of lands to produce pine and hardwood timber, livestock forage, and wildlife habitat for both game and nongame species; to ensure public safety on roads and other rights-of-way; to protect facilities and capital improvements; and to reduce hazardous fuel loads to protect resources from wildfire damage.

Silviculture operations designed to ensure the establishment of healthy stands, by altering species composition or density, are a major proposed program for herbicide use by the Forest Service. These operations include site preparation, hardwood and pine release, and precommercial thinning. Site preparation treatments are used to prepare newly harvested or inadequately stocked areas for a new stand of trees. Herbicides are used in site preparation to reduce the amount of undesirable vegetation available to compete with the desirable hardwoods or pines, while minimizing soil disturbance on the site. In the brown-and-burn method of site preparation, herbicides are used to reduce undesirable vegetation, to dry fuels, and to improve the effectiveness of a prescribed fire, thus enhancing planting and stand establishment. Release, precommercial thinning, and herbaceous weed control reduce competition, thereby improving the survival, growth, and health of desirable trees.

Right-of-way management operations include maintenance of roadsides, trails, power transmission and distribution lines, oil and gas pipelines, and railroad corridors. In roadside maintenance, vegetation is managed to prevent brush encroachment into driving lanes, to maintain visibility on curves, to permit drainage structures to function as intended, and to facilitate maintenance operations. Trails and utility corridors are also maintained for accessibility and safety.

Range improvement is done by the Forest Service to provide forage for domestic livestock by removing undesirable or noxious plant species and preparing range allotments for seeding by desirable forage plants. Noxious weeds are also controlled in other settings.

Wildlife habitat improvement activities include using herbicides to remove midstory and understory vegetation from pine stands managed for red-cockaded woodpeckers and other species, to release mast-producing hardwood trees, to control weed species in wildlife openings, and to maintain grassy openings free of weed and brush species.

TYPES OF APPLICATION METHODS AND HERBICIDE USAGE

The three basic types of herbicide application are (1) manual ground application, which requires hand-carried equipment; (2) mechanical ground application, which requires the use of truck- or tractor-mounted equipment; and (3) aerial application. Each is further categorized by the types of product or process it uses. Table 2-1 shows the number of acres in Region 8 treated annually with herbicides, by application method.

Herbicides currently being used are applied either as a spray (liquid formulations) or as granules (solid formulations). All types of spray application methods described here use systems designed to produce large droplets of herbicide, which minimize drift. The formulation of herbicides as granular products is intended to reduce drift because of the large size of granules. (Drift is described in more detail in section 4.) Figure 2-1 shows comparisons of the number of acres presently treated per year by each chemical and an estimate of the maximum number of acres that may be treated with each herbicide in future years.

Manual Ground Application Methods

Herbicide application by manual methods includes basal, soil spot, foliar (directed, herbaceous weed, and noxious weed), and cut-surface treatments. Manual ground application methods can be used in areas where a larger mechanical power unit is not practical or where a very selective treatment is desirable.

The number of workers involved in manual ground applications varies according to the project and type of activity. A manual spray applicator typically treats 0.1 to 0.4 ha (0.25 to 1 ac) per hour, depending on the density of vegetation, terrain, and equipment used.

Personnel applying herbicides may be exposed to herbicides and additives during mixing, loading, application, or cleanup operations. Inadvertent exposure may occur by direct or indirect contact with spray, a spill, or as a result of failed equipment, such as a disconnected or ruptured hose, a leaky gasket or washer, or a leaky nozzle.

Table 2-1

Number of acres treated annually with herbicides in Region 8 by application method

Application Method	Present		Maximum Anticipated[a]	
	Hectares	Acres	Hectares	Acres
Aerial[b]				
Foliar	729	1,800	4,251	10,500
Granular or pellet	729	1,800	3,036	7,500
Mechanical				
Foliar	4,332	10,700	13,320	32,900
Granular or pellet	2,632	6,500	3,563	8,800
Manual				
Granular or pellet	243	600	445	1,100
Foliar[c]	10,810	26,700	30,040	74,200
Basal bark/stem[d]	3,603	8,900	10,728	26,500
Soil treatment[e]	11,640	28,750	16,437	40,600
Cut surface[f]	9,960	24,600	11,296	27,900

[a] The numbers presented in this column are field estimates based on current herbicide use rates. They were made prior to the scoping process for two of the three EIS's to which this risk assessment will tier. They may not reflect alternatives proposed subsequently, but they are used as current best estimates for computation purposes only.

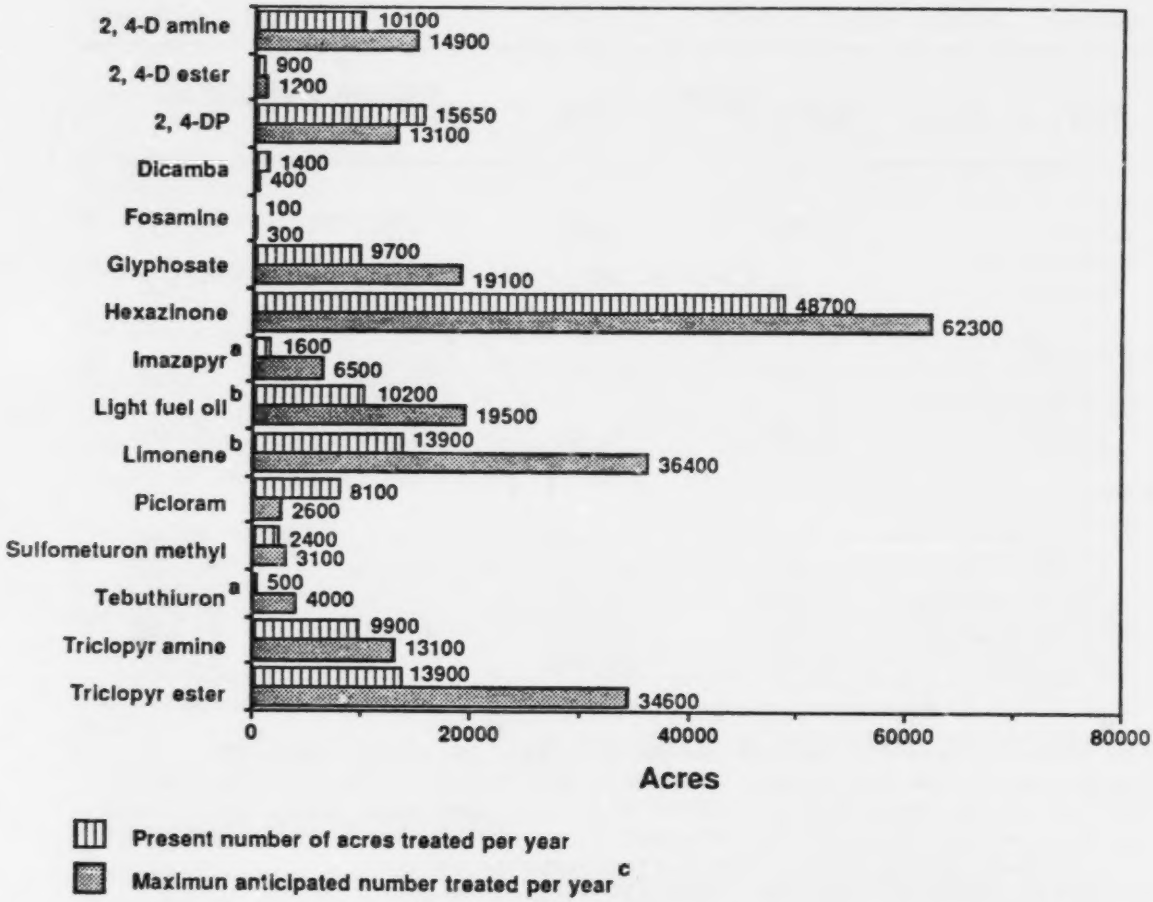
[b] Estimated potential use; not currently applied by air.

[c] Backpack/handsprayer; directed spray, herbaceous weed control, and so forth.

[d] Full or streamline treatments.

[e] Soil spot, basal soil, lacing, or streaking.

[f] Frill, injection, cut stump, hypohatchet, hack and squirt, and so forth.



[a] Projected use of herbicides that are currently not used in the R-8 program due to labeling.

[b] Used as a surfactant or penetrant, applied only in mixture with an herbicide.

[c] The numbers presented in this column are field estimates based on current herbicide use rates. They were made prior to the scoping process for two of the three EIS's to which this Risk Assessment will tier. They may not reflect alternatives proposed subsequently but are used as current best estimates for computation purposes only.

Figure 2-1--Current and projected number of acres to be treated annually by herbicides in Region 8

Basal Application

Basal application is used primarily for release, precommercial thinning, and right-of-way maintenance, though some site preparation work is done with this method. Two types of basal applications will be discussed: full basal and streamline treatments. In these treatments, herbicides are mixed with a liquid carrier, with or without additives, and are sprayed directly onto the bark of undesirable trees. Basal applications are generally made during the hardwood dormant season. The herbicide mixture is usually applied with a backpack sprayer and a spray gun or a spray wand. A backpack spray unit with a diaphragm pump is preferred over one with a piston pump because it is less likely to leak and it operates at a lower pressure.

Full basal treatments, which use a broad range of herbicides, are usually applied to stems up to 10 cm (4 in) in diameter. The lower 30 to 50 cm (12 to 20 in) of the stem are wet with herbicide mixture on all sides. While this method is no longer commonly used in Region 8, it is still the method of choice for some specialized projects.

Streamline treatments are generally applied to juvenile stems less than 8 cm (3 in) in diameter at breast height. The herbicide mixture is applied in a 3.8 to 5.1 cm (1.5 to 2 in) band to one side of the stem to juvenile bark near the base of the plant. Figure 2-2 shows how herbicides are applied using this method. Triclopyr ester mixed with limonene and diesel fuel is a common mixture applied by the streamline method.

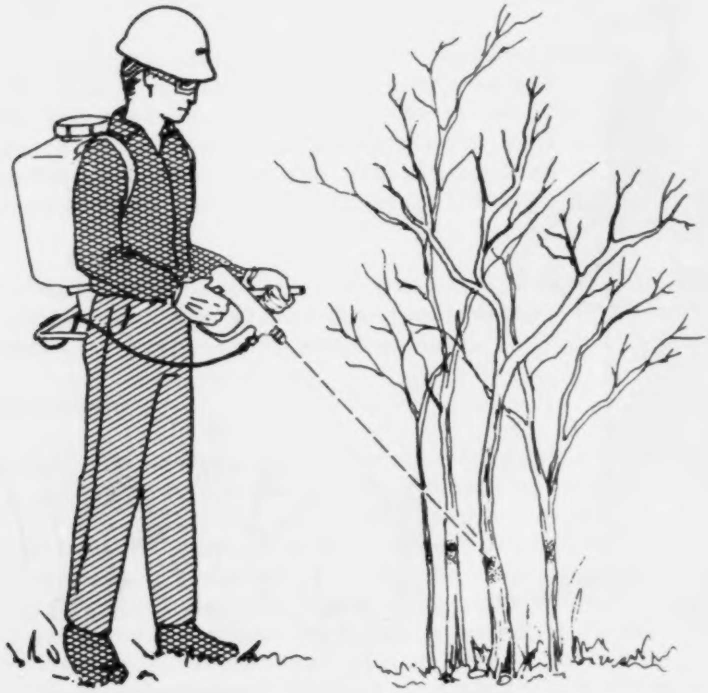


Figure 2-2--Applying herbicide using the streamline method

Soil Spot Applications

The soil spot application method is used for site preparation and release and, to a limited degree, for right-of-way maintenance. Applications can be made as either a spot grid (regular pattern) as individual stem treatment or in a pattern known as spot around. Formulated liquid herbicide is sprayed diluted or undiluted directly on the soil to control undesirable vegetation in the immediate area. Backpack sprayers equipped with a spray wand or spray gun are used. All sizes of vegetation can be treated using soil spot methods. However, one of the major factors in choosing both specific technique and spacing is the size of the target vegetation; larger targets require more spots.

Spot grid treatment is commonly used on sites with many stems per acre. Spots of herbicides are applied directly to the soil in a regular pattern. The dimensions of the grid are determined for each situation, based on the type of job, the kinds of vegetation to be controlled, the soil type on the site, and the like. Figure 2-3 illustrates this method of herbicide application.

Individual stem treatment is generally used on sites with fewer stems per acre. Herbicide is applied by directing the spray nozzle at the soil in the area where roots of the unwanted plants are growing.

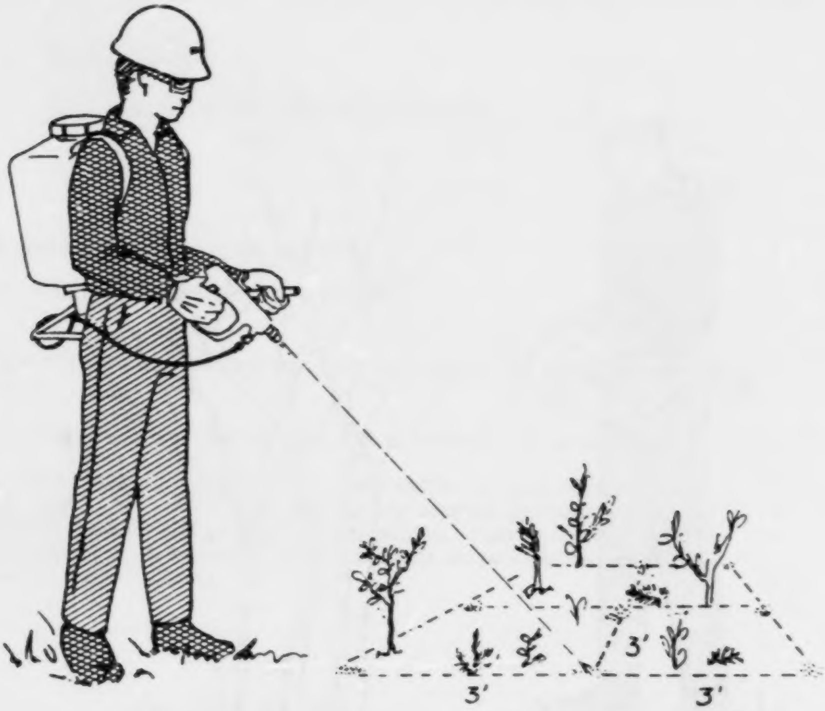


Figure 2-3--Applying herbicide using the spot-grid method

Spot around is a release method wherein spots are placed 1 m (3.3 ft) or more away from a young desired plant to reduce competition. This method is currently in limited use in Region 8.

Hexazinone is commonly applied as a soil spot application. Restrictions on use of this method based on soil type are found on several product labels.

Foliar Spray Applications

Foliar spray applications are used primarily to release first and second year pine stands. Two types are commonly distinguished: directed foliar application and herbaceous weed application. Liquid formulations are sprayed on the growing plants at different times depending on the technique being used. The timing is noted below for both types. Generally a backpack spray unit with a diaphragm pump and spray gun or wand is used to apply the herbicide.

Directed Foliar Spray Application. Directed foliar spray application is used to release young stands from competition less than 1.8 m (6 ft) tall. It is also used to reduce noxious or poisonous plant populations.

In this method, herbicide is sprayed in coarse droplets onto the foliage of undesirable plants and away from the foliage of desirable plants. Figure 2-4 shows an example of directed spray application. Herbicides can be applied as spring to fall treatments when target plants are fully leaved, green, and growing. Glyphosate, imazapyr, triclopyr, 2,4-D, and 2,4-DP can be used as directed foliar spray herbicides.

Herbaceous Weed Control. Herbaceous weed control involves the application of herbicide directly over the tops of desirable plants to control competing weeds and grasses. The herbicide is applied in a 1.2- to 1.5-m (4 to 5 ft) square or circle or in a continuous band that has desirable trees in the center. Herbicides are usually applied in the late winter or later in the spring when the competing vegetation is fully leaved and growing.

Some of the commonly used herbicides for herbaceous weed control are hexazinone, sulfometuron methyl, sulfometuron methyl + hexazinone, sulfometuron methyl + glyphosate, and imazapyr.

Cut-Surface Treatments

Cut-surface treatments are used to eliminate competing trees during site preparation, precommercial thinning, and release operations. Tree injection, frill or girdle, and cut-stump treatments are common types of cut-surface treatments. Currently only liquid herbicides are used for cut-surface treatments. These methods can be used throughout the year on virtually any size tree. However, some care must be taken to match timing with tree species to be treated. Various types of equipment are used for this method, including a hatchet and squirt bottle, a tubular tree injector, and injector-hatchets. Figure 2-5 shows one example of this method using a hatchet and a squirt bottle.

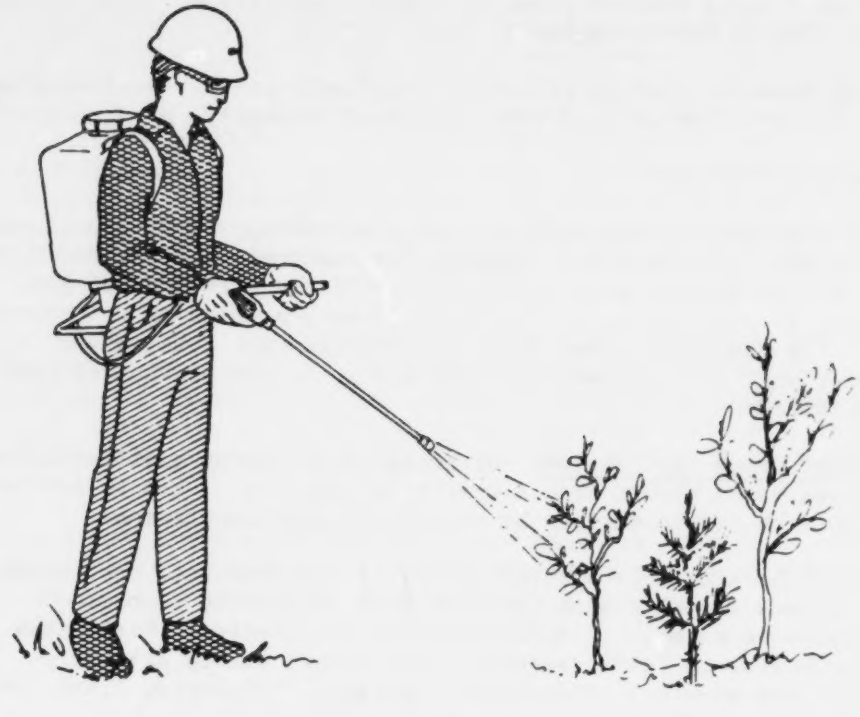


Figure 2-4--Applying herbicide using the directed spray method

Tree injection (in which the cambium of the target tree is exposed using a blade mounted on the tree injector and an herbicide solution is deposited in the cut) is most efficient on sites with sparsely distributed stems greater than 5 cm (2 in) in diameter at breast height. Some herbicides commonly used for injection are 2,4-D, triclopyr, picloram, imazapyr, and glyphosate.

The frill or girdle method involves cutting through the bark of a tree into the sapwood with an ax or hatchet. The cut surface is completely wet with herbicide. Wood chips produced during cutting are not removed, but are left to help hold the herbicide in the cut. Some herbicides commonly used for frill or girdle treatments are 2,4-D, triclopyr, picloram, imazapyr, and glyphosate.

The cut-stump treatment can be used on fresh or older stumps of any size. A pressurized backpack sprayer is used to thoroughly spray the cambial area (approximately the outer 2.5 cm (1 in)) of the stump. Herbicides used on cut stumps include 2,4-D, 2,4-DP, triclopyr, glyphosate, imazapyr, and picloram.

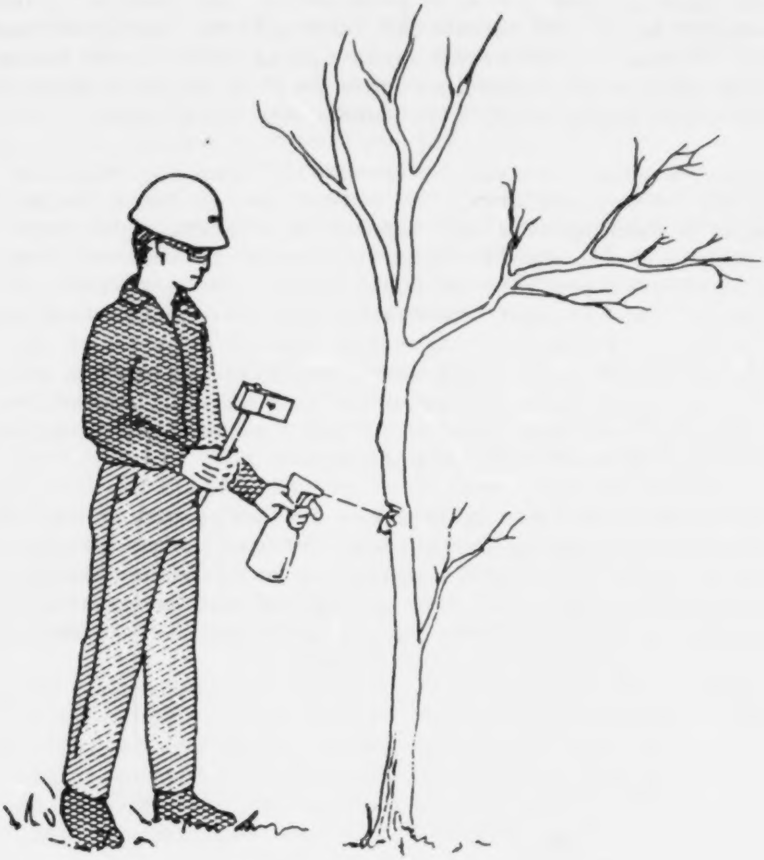


Figure 2-5--Applying herbicide using the hack and squirt method

Mechanical Ground Application Methods

Mechanical ground application methods are used in site preparation, release, and right-of-way corridor maintenance. They can be used in flat to rolling terrain. Mechanical application equipment includes tractors and trucks that have spray equipment or granule spreaders mounted on the vehicle. Both liquid and granular formulations are used. Application is broadcast, with some control being exercised by the operator (on/off, direction of application, timing, weather selection, etc.).

Granular herbicide applicators mounted on the rear of crawler tractors or skidders can be used for site preparation and conifer release. The unit can distribute the herbicide being carried on 2.4 to 6.1 ha (6 to 15 ac) in about 35 to 45 minutes. Hexazinone is the only granular herbicide currently being applied by this method; however, a granular formulation of imazapyr is expected to be used in forestry operations in the future.

Spray systems mounted on crawler tractors or skidders can be used to apply liquid herbicide formulations for site preparation and conifer release. These units normally apply 750 liters (l) (200 gallons (gal)) of herbicides in approximately 45 minutes, covering about 3.2 ha (8 ac). An example of this type of spray system is shown in figure 2-6. A variety of herbicides can be used, including hexazinone, glyphosate, and triclopyr.

Special truck-mounted spray systems are generally used for applying herbicides in right-of-way projects. A typical system has a large tank for the herbicide mix, a pumping-pressure regulating system, a lateral boom sprayer, and a nozzle head. Application is broadcast, although the operator has control over the rate of application, the direction of spray, timing of operation, and can shut the system off from inside the cab. The unit can apply 1,400 l (300 gal) in approximately 35 minutes at 16 kilometers per hour (km/hr) (10 miles per hour (mph)), covering about 5 ha (12 ac). All of the herbicides evaluated in this risk assessment are in common use for right-of-way application. Target vegetation is a major factor considered during herbicide selection.

Workers using these methods (generally a one or two person crew) may be exposed while mixing or applying herbicides. Mixer/loaders can be accidentally exposed as a result of a splash or spill of herbicide or a ruptured or disconnected hose. Drivers can be exposed to spray drift. Granule applicators are not likely to have significant herbicide exposures; exposure is restricted to small amounts of dust.

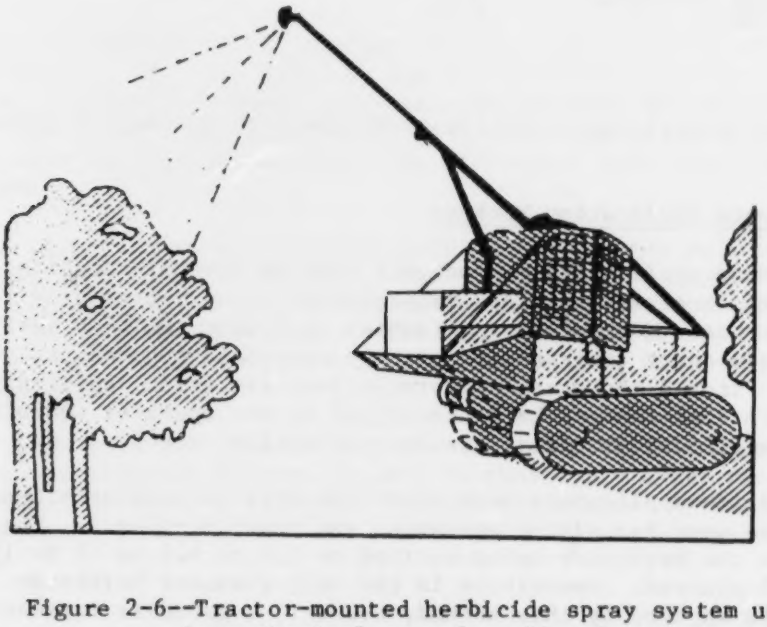


Figure 2-6--Tractor-mounted herbicide spray system used in mechanical ground applications

Aerial Methods

Aerial broadcast methods are particularly useful for covering remote managed areas, small scattered or isolated areas, or large forested areas in mountainous terrain. Aerial methods require relatively few people to treat areas. Vegetation condition, topography, and accessibility are less constraining for aerial methods than for other methods.

In the Southern Region, helicopters are expected to be the only vehicle used for aerial application of herbicides. Because helicopters are very maneuverable, the pilot can achieve good control of spray deposition and good drift control. Safety considerations, herbicide used, and application equipment determine the minimum altitude.

Herbicides are applied to target vegetation using specially designed spray nozzles and booms, granule applicators, and, where appropriate, drift-control adjuvants. These systems generally consist of a compressor or pressure source and a boom mounted across the aircraft, with nozzles spaced across the boom to distribute the herbicide solution evenly. The special design of these booms creates a minimum of air turbulence in the vicinity of the nozzle orifices, maintains a uniformly large droplet size, minimizing the production of aerosols. Figure 2-7 shows an example of a helicopter mounted with a spray system. Granular applicators are also specially designed to produce a uniform distribution of granules.

Depending on the purpose of the application, 16 to 40 contiguous ha (40 to 100 ac) can be covered in an hour of actual flying time (excluding refueling and loading time). Delivery rate will be 47 to 140 l/ha (5 to 15 gal/ac), again depending on the objective of the spray operation. Granular application is similar: 20 to 40 ha/hr (50 to 100 ac/hr), not including refueling and loading time and time required to move between sites.

Aerial application occurs only under favorable weather and terrain conditions. Some factors considered in planning aerial applications include: (1) wind speed and direction; (2) humidity and probability of rainfall; (3) temperature; (4) air temperature inversions; (5) terrain; and (6) sensitive areas within or adjacent to the spray area. Some of these factors are less constraining for aerial application of granules.

The number of workers involved in a typical aerial application project varies according to the type of activity. Some operations may require only 2 individuals, while others may need as many as 15 workers.

Hexazinone, glyphosate, imazapyr, sulfometuron methyl, triclopyr, and fosamine are expected to be the herbicides commonly used for aerial application. Tebuthiuron, 2,4-D, and 2,4-DP are also evaluated for aerial use.

PERSONAL PROTECTIVE EQUIPMENT

The type of clothing worn during an application operation is an important determinant of the exposure of workers. Specific protective clothing requirements may differ depending on the herbicide being applied.

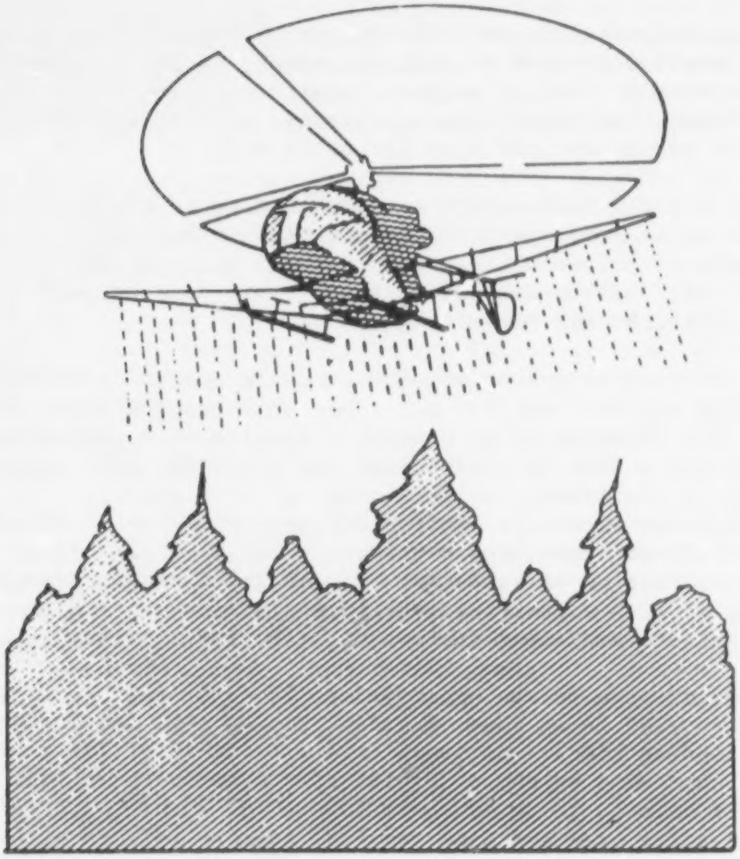


Figure 2-7--Aerial application using a helicopter with a mounted spray system

Herbicide labels and material safety data sheets indicate what type of protective clothing is necessary and when it is to be worn. Specific label requirements for protective clothing and equipment are followed.

For the person applying herbicides, minimal clothing requirements include a long-sleeved shirt and long pants that are made of tightly woven cloth and a hard hat with a plastic liner. Waterproof boots are worn when specified by the label. If leather boots are worn, they should be water-proofed with a good sealant. Each field crew carries a minimum of two eyewash bottles.

In addition to the minimal clothing requirements, some labels require respirators for persons loading a granular product into application equipment. Several labels specify either goggles or face shields, rubber gloves, and an apron for mixer/loaders of liquid products.

MITIGATION MEASURES

Measures intended to ensure the proper and safe application of herbicides on lands managed by the Forest Service in Region 8 are required by Federal, State, and regional regulations or laws. Federal and State laws and regulations set the minimum standards followed during herbicide application on forests and rangelands. Each Regional, Forest, or District Office may develop additional restrictions and precautions.

The Federal Insecticide, Fungicide, and Rodenticide Act requires pesticide manufacturers to register their chemicals with the U.S. Government (specifically, with the Environmental Protection Agency (EPA)) and list the allowable uses, application rates, and special restrictions on the herbicide's label. All of the herbicides considered in this risk assessment are registered for forestry application by EPA. Label rates, uses, and handling instructions are complied with according to Federal law.

The Department of Agriculture (Forest Service) has guidelines for herbicide application. Publications, such as Safety Training for Forestry Herbicide Applicators, Hand Application Methods for Commonly Used Forestry Herbicides in the South, and Certification Training: Applying Pesticides Correctly--A Self-Study Guide for USDA Forest Service Employees, provide additional guidelines for application of herbicides.

Mitigation measures, such as not spraying in sensitive areas, notifying the public, posting notice signs, and conducting water monitoring, are usually specified in site-specific vegetation management plans (called environmental assessments). Many mitigation measures developed for herbicide operations in the Southern Region are described in the environmental impact statements that this document supplements. Some specific examples include the following:

(1) Aerial spray application operations are suspended when wind velocity exceeds 9.6 km/h (6 mi/h) or inversion conditions exist.

(2) Weather conditions and spray delivery performance are monitored to minimize the chances of off-target drift, volatilization, runoff, or leaching of applied herbicides.

(3) Waterways and areas of open water are protected according to buffer strip requirements.

(4) Applications are made in strict conformance with herbicide label instructions, and applicators are supervised by a certified pesticide applicator.

(5) Protective clothing worn is consistent with herbicide labeling.

Section 3
HUMAN HEALTH HAZARD ANALYSIS

INTRODUCTION

This section presents the results of the human health hazard analysis. It includes a review of available toxicological information on the 11 herbicides, the surfactant limonene, and diesel oil and kerosene, classified as light fuel oils, that are being considered in the Forest Service vegetation management programs in the South (Region 8). The first subsection describes the sources of toxicity information used in the hazard analysis. The second subsection explains the laboratory toxicity testing terminology used to describe the toxic properties of the 14 herbicides and additives. The third subsection presents summaries of the threshold toxicity of each chemical drawn from the information that was available. The fourth and fifth subsections describe the potential for each of the 14 chemicals to cause genetic mutations and cancer respectively. The final subsection presents the details of the derivation of cancer potency for those chemicals suspected of being carcinogenic.

SOURCES OF TOXICITY INFORMATION

The toxicity of 2,4-D, 2,4-DP, dicamba, fosamine, glyphosate, hexazinone, picloram, tebuthiuron, and triclopyr to laboratory animals and humans is described in detail in the background statements of the Forest Service (Agricultural Handbook No. 633) (USDA, 1984, 1986). The toxicity of light fuel oil, imazapyr, and sulfometuron methyl is described in background statements written in conjunction with this risk assessment (Labat-Anderson, Inc. (LAI), 1986, 1987a,b). These documents are incorporated by reference into this risk assessment in accordance with 40 CFR 1502.16 and are available for review at all Forest Service supervisors' offices in Region 8, as well as the regional office. Little information exists concerning the toxicity of limonene. All information reviewed in the open literature is summarized in this hazard analysis.

Much of the data on pesticide toxicity have been generated to comply with the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended (7 U.S.C. 136 et seq.), which contains the established procedures for the registration, classification, and regulation of all pesticides, including herbicides. EPA is responsible for implementing FIFRA. Toxicity levels and related information from the series of studies submitted for registration are compiled by EPA in summary tables called "tox one-liners," which are available on request from EPA's Freedom of Information Office. EPA also has compiled and made available "science chapters" on dicamba, hexazinone, and picloram. A large body of additional toxicity information exists in the open literature, particularly for herbicides that have been used for many years.

The U.S. Department of Agriculture, Forest Service, funded an extensive literature search to ensure that all of the relevant available information was used in this risk analysis. A number of computerized literature retrieval data bases were searched to locate current literature pertaining to the carcinogenicity and mutagenicity of the herbicides, including Medline, the Embase (Excerpta Medica), Toxline, Hazardous Substances Data Base (HSDB), Registry of Toxic Effects of Chemical Substances (RTECS), the International Pharmaceutical Abstract data base, and the Chemical Carcinogenesis Research and Information System (CCRIS).

Data from the pesticide background statements (USDA, 1984, 1986; Labat-Anderson, 1986, 1987a,b) were reviewed and compared to summaries of studies submitted to the Environmental Protection Agency for the registration of the herbicides and additives. When possible, studies that have been reviewed and validated by EPA were used to set toxicity reference levels. No studies were used that have been invalidated by EPA.

HAZARD ANALYSIS TERMINOLOGY

Because of the obvious limitations of testing chemicals on humans, judgments about potential hazards are based on the results of toxicity tests on laboratory animals. Toxicity test results are supplemented by available information, if any, on actual human poisoning incidents and effects on human populations. The discussion of laboratory toxicity testing that follows is drawn from Hayes (1982), Doull et al. (1980), Environ Corporation (1985), and Loomis (1978).

Laboratory Toxicity Testing

Test Animal Species

Laboratory test animals serve as models that indicate the possible effects of a pesticide in humans. The ideal test animal would metabolize a compound in the same manner as a human and have the same susceptible organ systems. Results of such tests could then be directly extrapolated to humans, making some adjustment for differences in body weight and body surface area. Although no test animal has proved ideal, species such as rats, mice, rabbits, hamsters, guinea pigs, dogs, and monkeys have proved to be consistent indicators for certain types of toxicity tests, routes of administration, and types of chemicals. Rats and mice are the most commonly used animals because of their low cost, relative ease of handling, documentation of genetic background, documentation of susceptibility to disease, and relatively short life span (2 to 3 years).

Toxic Endpoints and Toxicity Reference Levels

Toxicity tests are designed to allow the accurate evaluation of specific herbicidal toxicological properties such as specific toxic endpoints (for example, temporary or chronic debilitation, carcinogenicity, or fatality) and toxicity reference levels (for example, no-observed-effect levels (NOEL)). In addition to the type of test animal used, variables of toxicity tests include test duration, route of administration, dose levels, dosing schedule, number of test groups, number of animals per group, and other individual specific variables (for example, sex and age). Toxicity tests also vary on the basis of the assumption of whether the effect in question is a threshold effect or a nonthreshold effect.

Threshold and Nonthreshold Effects

Most chemicals are assumed to have a threshold level of toxic effects on a local basis (at the site of administration) or a systemic basis (acting throughout the body), below which no adverse effects occur to the test organism. In animal testing, when the effect threshold is exceeded, systemic effects, such as liver or kidney damage or dysfunction, weight loss, or reproductive impairment, may occur. A no-observed-effect level, the dose where none of these effects is evident, and a lowest-effect level (LEL) are the dose levels that bracket the threshold of effects. Chemicals are generally assumed to possess no such threshold level for cancer and mutations. Thus, these toxic endpoints may occur (with a certain level of probability) even in the presence of extremely small quantities of the substance. This is a controversial issue, however, and although data supporting the evidence of thresholds exist for some chemicals, regulatory authorities generally take the more conservative approach, which assumes no thresholds for mutagenicity and carcinogenicity.

In this hazard analysis, threshold effects are discussed first. The nonthreshold effects, mutagenicity and cancer, are discussed in the last two subsections.

Duration of Toxicity Tests

The duration of toxicity tests ranges from very short-term acute tests to longer subchronic studies to chronic studies that may last the lifetime of an animal. Acute toxicity studies involve administration of a "single" dose to each member of a test group (either at one time or in a cumulative series over a short period of less than 24 hours). Subchronic toxicity studies, used to determine the effects of multiple doses, usually last from a few days to 3 months (3 to 90 days), but generally less than half the lifetime of the test animal. Chronic studies, also used to determine the effects of multiple or continuous doses, normally last 1 to 2 years but generally more than half the test species' lifetime. Studies may be designed to evaluate both chronic toxicity and oncogenicity. These studies are conducted over the major portion of the test organisms' lifetime; usually 18 to 24 months for mice and rats.

Routes of Administration

Routes of administration include oral (by gavage [forced into the stomach through a tube] or fed in the diet), dermal (applied to the skin), inhalation (through exposure to vapors or aerosol particles), and parenteral (injection other than into the intestine). Parenteral routes include subcutaneous (injected under the skin), intraperitoneal (injected into the abdominal cavity), and intravenous (injected into a vein). Oral, dermal, and inhalation doses most closely duplicate the likely routes of exposure to humans. Parenteral doses are used in testing drugs but are not widely used in toxicity pesticide testing because they bypass the test animal's natural protective mechanisms (including barriers such as the skin, lung surface, and the surface of the digestive tract).

Dosing Levels

Doses are expressed in several ways: as milligrams (mg, which is 1/1,000 of a gram) of the chemical per kilogram (kg, which is 1,000 grams) of body weight of the test animal, in parts per million (ppm) in the animal's diet, or in milligrams per liter (mg/l) in the air the animal breathes or in the water the animal drinks.

Dosing in long-term studies is generally done through the diet with specified amounts in parts per million in the food. The known weight of the test animals over the test period and the amount of food actually consumed are used to convert ppm in the diet to milligrams of chemical per kilogram of body weight per day (mg/kg/day) for extrapolation to humans. In general, at least three dosing levels are used in addition to the zero dose given a control group. Usually, animals of each sex are dosed in groups of 8 to 50.

For the discussion that follows of toxicological studies of the herbicides and additives being considered for use in Region 8, doses reported in parts per million have been converted to mg/kg/day using the following conversion factors: mouse 1 ppm = 0.15 mg/kg/day; rat 1 ppm = 0.05 mg/kg/day; rabbit 1 ppm = 0.03 mg/kg/day; and dog 1 ppm = 0.025 mg/kg/day (USDA, 1984).

Types of Toxicity Studies

Acute Toxicity Studies

Acute toxicity studies are used to determine the toxicity reference level known as the median lethal dose (LD_{50}), which is the dose that kills 50 percent of the test animals. The lower the LD_{50}, the greater the toxicity of the chemical. The LD_{50} ranges and toxicity categories used in this risk assessment are those of the EPA classification system using rat oral LD_{50}'s, as shown in table 3-1, adapted from Walstad and Dost (1984). Categories of toxicity using this classification system include: severe (rat LD_{50} less than 50 mg/kg), moderate (rat LD_{50} 50 to 500 mg/kg), slight (rat LD_{50} 500 to 5,000 mg/kg), and very slight (rat LD_{50} 5,000 to 50,000 mg/kg).

Common routes of exposure for acute toxicity studies include oral, dermal, and inhalation, which are the most common exposure routes in real-life situations. Because lethality is the intended toxic endpoint, dose levels usually are set relatively high in acute studies. Toxic symptoms displayed by the animals may be recorded throughout the study, and tissues and organs are examined for abnormalities at the end of the test. The animals most commonly used for oral LD_{50}'s are the rat and the mouse because they are economical, readily available, easy to handle, and they are similar to humans in their response to chemicals. In addition, much toxicological data already exist for these species, which facilitates comparison with toxicity data developed for other chemicals. Rabbits are used most often to determine dermal LD_{50}'s because they have greater dermal sensitivity than many other animals.

Table 3-1

Acute toxicity classification and acute toxicities of the 14 herbicides and additives being evaluated for use in vegetation management in relation to other chemicals

Toxicity Category[a] (label signal words)	Herbicide or Other Chemical Substance	Oral LD_{50} for Rats (mg/kg)	Equivalent Human Dose
IV Very slight		5,000 - 50,000 (range)	More than 1 pint
	Sugar	30,000	
	Kerosene	28,000	
	Fosamine	24,400	
	Ethyl alcohol	13,700	
	Picloram	8,200	
	Diesel Oil	7,380	
	Imazapyr	>5,000	
	Sulfometuron Methyl	>5,000	
	Limonene	5,000	
III Slight (caution)		500 - 5,000 (range)	1 ounce to 1 pint
	Glyphosate	4,320	
	Table salt	3,750	
	Bleach	2,000	
	Aspirin, Vitamin B_3	1,700	
	Hexazinone	1,690	
	Formaldehyde	800	
	Dicamba	757	
	Tebuthiuron	644	
	Triclopyr	630	
	2,4-DP	532	
II Moderate (warning)		50 - 500 (range)	1 teaspoon to 1 ounce
	2,4-D	375	
	Malathion (insecticide)	370	
	Carbaryl (insecticide)	270	
	Caffeine	200	
	Paraquat (herbicide)	95	
I Severe (danger - poison)		0 - 50 (range)	1 teaspoon or less
	Nicotine	50	
	Strychnine (rodenticide)	30	
	Parathion (insecticide)	13	
	TCDD (a dioxin)	0.1	
	Botulinus Toxin	0.00001	

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[a] Categories, signal words, and LD_{50} ranges are based on a classification system used by EPA for labeling pesticides.

Source: Maxwell (1982) (as cited in Walstad and Dost (1984).

Because death represents the extreme toxic consequence for judging possible effects from the use of pesticides, the policies of regulating agencies regarding acceptable intake levels of these chemical compounds most often are not based on acute studies. Rather, they are based on toxicity tests designed to find the dose level that produces no effects despite repeated exposures over an extended period of time in the animal species tested. Figure 3-1 illustrates the relationship between the LD_{50} and the no-effect level.

Acute dermal, primary dermal, dermal sensitization, and primary eye irritation tests assess additional acute hazards of a chemical. Albino rabbits, which are used in these studies, are typically more sensitive to these tests than other test mammals and humans. Thus, the chance of obtaining false negative test results is reduced. The acute dermal test enables the researcher to determine an LD_{50} value for the test chemical. Rabbits are exposed to the test chemical for a 24-hour period. Observations of the adverse effects (erythema and edema) of the chemical are made using the Draize scoring system (Draize et al. 1944). For the primary dermal test, a constant dosage level of 0.5 ml or 0.5 g is used. Observations for this test are made over a longer period of time than the acute dermal study (normally 72 hours).

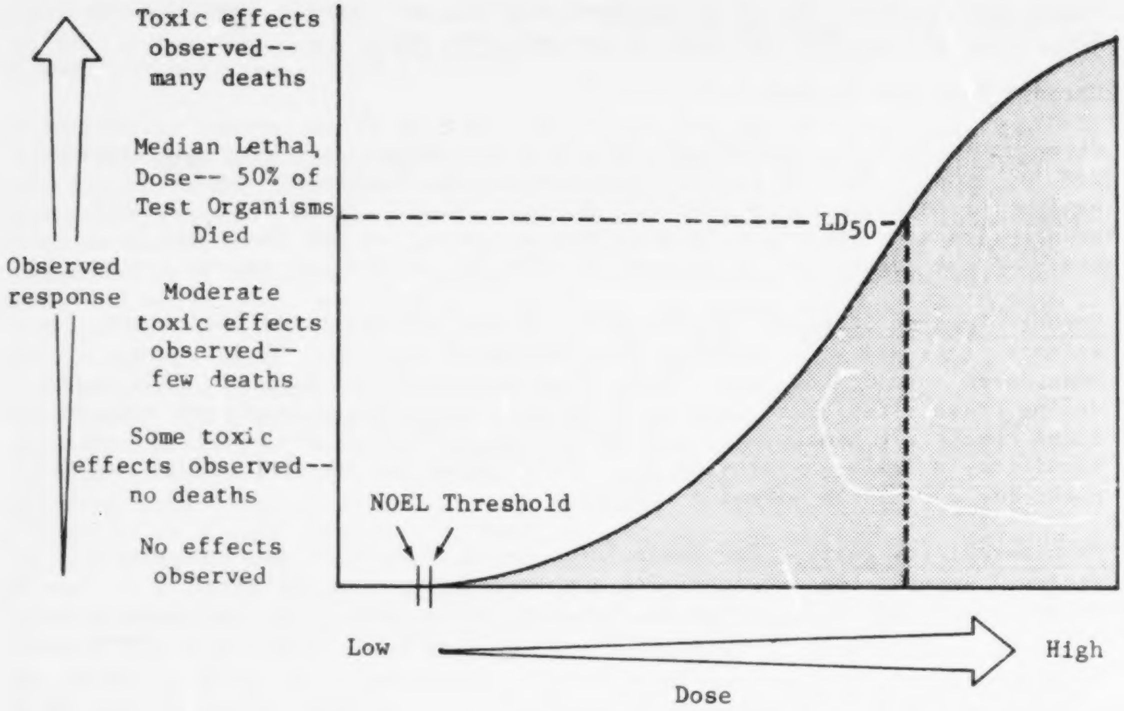
The dermal sensitization test uses guinea pigs, which respond much like humans to chemicals. This test measures the ability of the animal to invoke an immune response to successive chemical exposure. The primary eye irritation test measures the toxicological effect of a chemical to the eye (the cornea in particular). The damage a chemical may cause to the ability of the cornea to transmit light (corneal opacity) is an important result of this test.

EPA classifies chemicals in one of four toxicity categories based on the effect a chemical has on the cornea and the skin (EPA, 1974) (table 3-3 in a later section).

Subchronic Toxicity Studies

Subchronic studies are used to determine the toxicity reference level, called the no-observed-effect level (NOEL), which is the highest dose level at which no toxic effects are observed. If a chemical produces effects at the lowest dose tested (LDT) in a study, the NOEL must be at some lower dose. If the chemical produces no effects, even at the highest dose tested (HDT), the NOEL is greater than the HDT. Another toxic endpoint of interest is the lowest dose showing toxic effects, the lowest effect level (LEL). For local and systemic effects, the chemical's threshold of effect lies between the NOEL and LEL for the tested species. (See figure 3-1.)

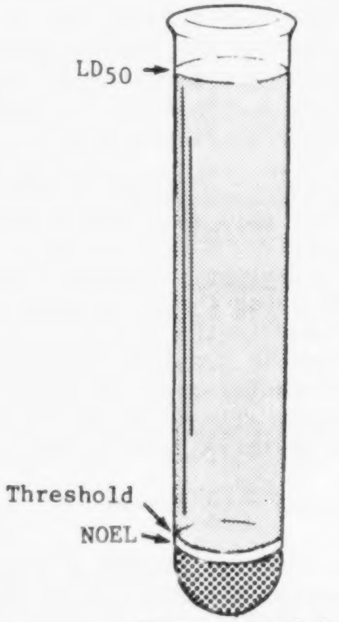
Subchronic studies, normally using lower dose levels than acute studies, provide information about systemic effects, cumulative toxicity, the latency period (the time between exposure and the manifestation of a toxic effect), the reversibility of toxic effects, and appropriate dose ranges to use in chronic tests. The adverse effects may include decreased rate of food consumption, change in body weight, altered enzyme levels, changes in



LD$_{50}$ - Acute lethal dose. One-time or short-term dose that is lethal to 50 percent of treated animals.

Threshold - Dose level at which toxic effects are first observed in test animals.

NOEL - No-observed-effect level. Long-term dose that does not result in apparent adverse effects in test animals.



(Not to Scale)

Figure 3-1--Relationships among toxicity reference levels blood constituents (red or white blood cells), undesirable constituents in the urine, microscopic changes in tissues, and death.

Chronic Toxicity Studies

Chronic studies, like subchronic studies, can be used to determine systemic NOEL's. NOEL's derived from chronic toxicology studies are useful in evaluating safe long-term chemical exposure levels for humans. Chronic studies are also important in determining doses that are hazardous to reproductive success or in determining whether a chemical causes cancer.

Chronic Feeding Studies. In general, for the shorter-lived laboratory animals, rats and mice, feeding experiments of more than 90 days are considered chronic studies. These tests can determine systemic NOEL's and define organ sites where long-term exposure can cause deleterious effects. Blood chemistry, hematology, histopathology, and gross pathology of the laboratory animals can provide detailed information about the effect of the pesticide during the animal's lifetime.

Carcinogenicity Tests. Carcinogenicity tests (cancer or oncogenicity studies) examine the potential for a chemical to cause malignant or nonmalignant tumors or leukemias when fed in the diet over the animal's lifetime. Testing is normally conducted with rats or mice for a 2-year period.

Teratogenicity Tests. Teratogenicity tests, now termed developmental studies, are used to determine the potential of a chemical to cause malformations in an embryo or a developing fetus between the time of conception and birth. These studies generally use pregnant female rats or rabbits dosed during the early and middle period of gestation while the organs of the fetus are developing. The animals are monitored for structural deformities and occasionally for functional disorders.

Reproduction Studies. Reproduction studies are conducted to determine the effect of a chemical on reproductive success, which is indicated by fertility, direct toxicity to the developing fetus, and survival and weight of offspring. Usually, the test animals are rats and lower doses are used than those for teratogenicity studies. Both male and female rats are exposed to the chemical for a number of weeks before mating. The number of resulting pregnancies, stillbirths, and live births are recorded. Histopathological evaluations of the reproductive organs of parents, and occasionally of selected pups, is conducted. Tests may be conducted over two or three generations.

Mutagenicity Assays

Mutagenicity assays are used to determine the ability of a chemical to cause physical changes (mutations) in the basic genetic material (deoxyribonucleic acid (DNA)), especially changes that could be passed on from one generation to the next. The species used in these tests range from primitive organisms, such as the bacteria Salmonella spp., Escherichia spp., and Streptomyces spp.; the mold Aspergillus spp.; the yeast Saccharomyces spp.; and the fruitfly Drosophila spp., to the more advanced organisms that include mammalian species. Tests may be conducted *in vivo* (within the body of the living organism) or *in vitro* (on cells cultured outside the body in a petri dish or test tube).

Mutagenicity assays can be divided into three categories: (1) tests for detecting gene mutations, (2) tests for detecting chromosomal aberrations, and (3) tests for detecting primary DNA damage. Included in the first group are microbial assays, involving prokaryotic microorganisms (organisms such as bacteria and cyanobacteria that lack a nucleus separated from the cytoplasm by a membrane) and eukaryotic microorganisms (organisms with a well-defined nucleus enclosed in a membrane, including all nonprokaryotes such as yeasts, other fungi, and mammals) developed to detect reverse mutations (a mutant gene that mutates back to the wild type) and to a limited extent, forward mutations (a wild type gene that undergoes mutation). Because many mutagens are inactive before bioactivation (by metabolic activity), bacterial tests may include a bioactivation system, such as an S9-fraction, which consists of microsomal enzymes of rats' or other animals' livers to activate the mutagen. A host-mediated assay is conducted to detect mutagenic effects in a microorganism, such as bacteria, by injecting it into the peritoneal cavity of the host (usually mice) to allow for a better bioactivation environment of the mutagen *in vivo*. Other tests useful for predicting gene mutations are the fruitfly sex-linked recessive lethal test, which measures the frequency of lethal mutations, the mouse specific locus test, which detects mutagenicity in germ cells *in vivo*, and mammalian somatic cell assays *in vitro* using mouse lymphoma cells, human lymphoblasts, and Chinese hamster ovary cells to detect forward and reverse mutation.

Examples of tests for detecting chromosomal effects include mammalian cytogenetic assays in Chinese hamster ovary cells *in vitro* and mice bone marrow micronucleus *in vivo*. The dominant lethal test in rodents, which determines lethal mutation in germ cells, and the heritable translocation test in mice, which detects the heritability of chromosomal damages, are both important tests performed with live animals. Fruitflies and other insects also are used to detect heritable chromosomal effects *in vivo*.

The existence of DNA damage caused by mutagens is detected by biologic processes, such as DNA repair and recombination, which occur after DNA damage. Tests to determine such processes use bacteria, yeast, and mammalian cells *in vitro*, with or without metabolic activation. Unscheduled DNA synthesis, for example, is often used to indicate DNA repair in human cells *in vitro*. Mitotic recombination and gene conversion indicate DNA damage in yeast, and sister chromatid exchange indicates DNA damage in mouse lymphoma cells, Chinese hamster ovary cells, and human lymphocytes.

The methodology for testing and evaluating results for mutagenicity studies (for example, battery of tests, weighted evidence) will be discussed in the mutagencity section later in this section.

THRESHOLD TOXICITY OF THE 14 HERBICIDES AND ADDITIVES

The toxicity reference levels used in this risk assessment to describe both acute and chronic threshold effects of the 14 herbicides and additives considered for use in Region 8 are presented in table 3-2. The table gives two types of NOEL's. The first NOEL listed is for general systemic effects, such as growth retardation, decreased red blood cell counts, and liver and kidney effects. Most of the systemic NOEL's take into account EPA-validated 2-year chronic feeding studies. For fosamine, picloram, and triclopyr, subchronic study NOEL's were used because they are the lowest NOEL's found in the literature. The other type of NOEL given is the lowest for reproductive effects, including infertility, general maternal and fetal toxicity, and birth defects (teratogenesis). Where information is available, NOEL's are given for both reproductive and teratogenic effects. Reproductive and teratogenic NOEL's are considered separately from general systemic NOEL's because, in some cases, mammals have been shown to be particularly vulnerable to the toxic effects of chemicals during reproduction and development. All the NOEL's used are the lowest found in EPA-validated studies.

There are many possible reasons for studies not to be validated by EPA; for example, insufficient sample size or incomplete description of the study methodology. Some of these studies, however, still provide useful information on toxic effects. Results of acute dermal, primary dermal, primary eye, and subchronic dermal studies are found in table 3-3.

The following subsections summarize the most relevant acute and chronic toxicity tests that have been conducted on the 14 herbicides and additives. Areas where no validated studies exist or in which EPA has requested additional studies are noted.

2,4-D

2,4-D is classified as moderately toxic (see table 3-1) in mammals with an LD_{50} in rats of 375 mg/kg (EPA, 1986b). Symptoms of toxicity observed in humans after ingestion of 2,4-D include irritation to the gastrointestinal tract, chest pains, and muscle twitching (USDA, 1984). Excessive dermal contact with 2,4-D in humans causes skin irritation, tingling of extremities, nausea and vomiting, and muscle aches and loss of function (USDA, 1984). Prolonged breathing of 2,4-D vapors causes coughing, burning, dizziness, and temporary loss of muscle coordination (USDA, 1984). Even though dermal absorption of 2,4-D is limited, the herbicide has been reported to produce peripheral neuropathy (characterized by progressive numbness, aching of the extremities, and eventually paralysis) in a few individuals after accidental acute exposure (Goldstein et al., 1959). In several cases, the recovery has not been complete. These effects have not been produced in laboratory animals. Rats exposed dermally to 12- and 24-percent solutions of 2,4-D amine for up to 3 weeks exhibited no signs of peripheral neuropathy, although skin lesions (ulcerative dermatitis), decreased body weights, and increased kidney weights were observed (Mattsson et al., 1986a,b; EPA, 1986a). 2,4-D was irritating to rabbit skin in a primary dermal and acute dermal study and

Table 3-2

Laboratory-determined toxicity levels used in the risk analysis

Chemical	Lowest Acute Oral LD_{50} in Rats	Lowest Systemic NOEL	Lowest Reproductive, Maternal or Fetotoxic, and/or Teratogenic NOEL
2,4-D[a]	375 mg/kg (EPA, 1986a)	1.0 mg/kg/day, 1-year tentative NOEL for 2-year rat feeding study (EPA, 1986b)	Fetotoxic and maternal NOEL = 5 mg/kg/day, rat reproduction study (EPA, 1986a)
2,4-DP[a]	532 mg/kg, rat (EPA, 1984a)	5 mg/kg/day, 90-day rat feeding study (EPA, 1984a)	Three-generation rat reproduction study, NOEL = 6.25 mg/kg/day (EPA, 1984a)
Dicamba[a]	757 mg/kg (USDA, 1984)	15.8 mg/kg/day, 15-week rat feeding study (EPA, 1987e)	Fetotoxic and maternal NOEL = 3.0 mg/kg/day rabbit teratology study (EPA, 1983a)
Fosamine[b]	24,400 mg/kg (EPA, 1987b)	25 mg/kg/day, 6-month dog feeding study (Schneider and Kaplan, 1983, in USDA, 1984)	Teratogenic NOEL = 1,000 ppm (50 mg/kg/day), rat teratology study (CDFA, 1986)
Glyphosate	4,320 mg/kg (EPA, 1986c)	Greater than 31 mg/kg/day, 26-month rat feeding study (EPA, 1986c)	Fetotoxic NOEL = 10 mg/kg/day, 3-generation rat reproduction study (EPA, 1986c)
Hexazinone	1,690 mg/kg (EPA, 1986d)	10 mg/kg/day, 2-year rat feeding/oncogenic study (EPA, 1986d)	Fetotoxic NOEL = 50 mg/kg/day, 3-generation rat reproduction study (EPA, 1982a)

Table 3-2 (continued)

Laboratory-determined toxicity levels used in the risk analysis

Chemical	Lowest Acute Oral LD_{50} in Rats	Lowest Systemic NOEL	Lowest Reproductive, Maternal or Fetotoxic, and/or Teratogenic NOEL
Imazapyr[b]	Greater than 5,000 mg/kg (EPA, 1985b)	500 mg/kg/day (HDT), 90-day rat feeding study (American Cyanamid, 1985a)	Maternal toxic NOEL = 300 mg/kg/day, rat teratology study (EPA, 1985b)
Light fuel oils[b]	Diesel oil-- 7,380 mg/kg (Beck et al., 1982)	Diesel oil-- 7.38 mg/kg/day based on LD_{50}/1000	Diesel oil-- no teratogenic effects at 100 or 400 ppm, rat inhalation teratology study (Mecler and Beliles, 1979) equivalent oral dose 751 mg/kg/day (NRC, 1983)
	Kerosene--greater than 28,000 mg/kg (HSDB, 1987a)	Kerosene--28 mg/kg/day based on LD_{50}/1000	Kerosene--751 mg/kg/day based on diesel oil NOEL
Limonene[b]	5,000 mg/kg (HSDB, 1987b)	Less than 227 mg/kg/day based on slight decrease in body weight in a rat feeding study (Tsuji et al., 1975, as cited in HDSB, 1987b)	Teratogenic NOEL less than 2,363 mg/kg/day, based on fetal bone formation in a mouse teratology study (Kodama et al., 1977, as cited in HSDB, 1987b). Set at 227 mg/kg/day, based on systemic NOEL.
Picloram[a]	8,200 mg/kg, rat (EPA, 1984c)	7 mg/kg/day, 6-month dog feeding study (Mullison, 1985)	Reproductive NOEL = 50 mg/kg/day, 3-generation rat study (EPA, 1988)
Sulfometuron methyl	Greater than 5,000 mg/kg (EPA, 1984d)	2.5 mg/kg/day, 2-year rat feeding study (DuPont, 1986)	Reproductive NOEL = 25 mg/kg/day, 2-generation rat reproduction study (DuPont, 1986)

Table 3-2 (continued)

Laboratory-determined toxicity levels used in the risk analysis

Chemical	Lowest Acute Oral LD_{50} in Rats	Lowest Systemic NOEL	Lowest Reproductive, Maternal or Fetotoxic, and/or Teratogenic NOEL
Tebuthiuron[b]	388 - 478 mg/kg[c]	12.5 mg/kg/day, 3-month dog feeding study (EPA, 1984e)	Reproductive NOEL = 5.0 mg/kg/day, 2-generation rat study (EPA, 1987f)
Triclopyr[a]	630 mg/kg (EPA, 1986f)	2.5 mg/kg/day (HDT), 6-month dog feeding study (40 CFR Part 180, 50 (84):184-85, May 1, 1985)	Fetotoxic NOEL less than 10 mg/kg/day, rabbit teratology study (EPA, 1986f)

[a] A 2-year rat, mouse, or dog feeding study established a higher systemic NOEL but the lower subchronic NOEL was used.

[b] No valid chronic studies available.

[c] Elanco. 1989. Information in letter written in response to Draft EIS.

Conversion Factors:
- mouse 1 ppm = 0.150 mg/kg/day
- rat (lifetime) 1 ppm = 0.05 mg/kg/day
- rabbit 1 ppm = 0.030 mg/kg/day
- dog 1 ppm = 0.025 mg/kg/day

Source: USDA, 1984.

Table 3-3

Dermal toxicology studies of the 14 herbicides and additives

	Acute Dermal	Primary Dermal	Primary Eye	Subchronic Dermal
2,4-D	III[a], $LD_{50} > 3,980$ mg/kg, 21.1% 2,4-D, rabbits tested (EPA, 1986b)	III, 21.1% 2,4-D, rabbits tested (EPA, 1986b)	I, rabbits tested (EPA, 1986b)	LD_{50} up to 3,980 mg/kg (HDT = 3,980 mg/kg), local skin inflammation, amines and ester formulation, 21-day dermal test on rabbits (USDA, 1984)
2,4-DP	III, $LD_{50} > 2$g/kg (ODT) mild erythema, complete recovery in 24-hours, rabbits tested (EPA, 1984a)	IV, Weedone (a.i., 2,4-DP), rabbits tested (EPA, 1984a)	IV, rabbits tested (EPA, 1984a)	NA[b]
Dicamba	III, $LD_{50} > 2,000$ mg/kg (ODT), DMA salt, rabbits tested (EPA, 1986g)	IV, rat tested; rabbits tested (EPA, 1986g)	III, rabbits tested (EPA, 1986g)	Slight dermal irritation and edema at a 100 mg/kg/day dosage level, moderate dermal irritation and edema at a 2,500 mg/kg/day dosage level, rabbit 21-day dermal test (EPA, 1986g)
Fosamine	II, $LD_{50} > 1,683$ mg/kg, formulation 43% a.i. and 69% reaction products, rabbits tested (EPA, 1987a)	IV, 43% a.i. and 69% reaction product Na-lignosulfonte 0.02%, guinea pigs tested (EPA, 1987a)	NA	No reaction at 0, 50% and 100% dilutions; formulation 43% a.i. and 69% reaction-product; rabbits tested (EPA, 1987a)
Glyphosate	III, LD_{50} (F) $\geq 7,940$ mg/kg, LD_{50} (M) $\geq$ 5,010 mg/kg, rabbits tested (EPA, 1986c)	IV, rabbits tested (EPA, 1986c)	III, rabbits tested (EPA, 1986c)	NOEL = 1,000 mg/kg/day LEL= 5,000 mg/kg/day Slight erythema, and edema; 21-day dermal test of rabbits (EPA,1986c)
Hexazinone	III, $LD_{50} > 5,278$ mg/kg, rabbits tested (EPA, 1986d)	III, rabbits tested (EPA, 1982a)	II, 94% a.i. (EPA, 1986d)	NA

Table 3-3 (continued)

Dermal toxicology studies of the 14 herbicides and additives

	Acute Dermal	Primary Dermal	Primary Eye	Subchronic Dermal
Imazapyr	III, $LD_{50} > 2,000$ mg/kg, rabbits tested; $LD_{50} > 2,000$ mg/kg, rats tested (EPA, 1985b; American Cyanamid Co., 1985b)	IV, rabbits tested (EPA, 1985b)	III, rabbits tested (EPA, 1985b)	NOEL = 400 mg/kg/day (HDT), 21-day dermal test on rabbits (EPA, 1985b)
Light Fuel Oil	Diesel oil--III (tentative), 6 ml/kg (ODT), rabbits tested (Beck et al., 1982)	Diesel oil--II (Beck et al., 1982)	Diesel oil--IV (Beck et al., 1982)	Diesel oil--avg. weight loss of 0.38 kg/animal, no mortality; 3,280 mg/kg dosage level; 21-day dermal test of rabbits (Beck et al., 1982)
	Kerosene (Jet Fuel A)--III (tentative), $LD_{50} > 5,000$ mg/kg, rats tested (Beck et al., 1982)	Kerosene (Jet Fuel A)--IV, rabbits tested; IV, guinea pigs tested (Beck et al., 1982)	Kerosene (Jet Fuel A)--IV, rabbits tested; IV, guinea pigs tested (Beck et al., 1982)	Kerosene (Jet Fuel A--) 75% mortality (severe dermal irritation, anorexia, weight loss, depression, and pale liver and kidneys) at 6400 mg/kg/day dosage level; 21-day test of rabbits (Beck et al., 1982)
Limonene	III (tentative), $LD_{50} > 5$ g/kg, Cide-Kick formulation, rats tested (SCM Organic Chem., 1981)	IV (SCM Organic Chem., 1981)	NA	NA
Picloram	III, $LD_{50} > 4$ g/kg (HDT), rabbits tested (EPA, 1984c)	IV (tech. washed) rabbits tested (EPA, 1984c)	III, rabbits tested (EPA, 1984c)	NA
Sulfometuron Methyl	III, $LD_{50} > 2,000$ mg/kg, rabbits tested (EPA, 1984d)	III, rabbits tested; IV, guinea pigs tested 75% a.i. (EPA, 1984d)	III, 75% a.i. (EPA, 1984d)	NOEL > 2,000 mg/kg/day (HDT) OUST formulation, 21-day dermal rabbit test (EPA, 1984d)
Tebuthiuron	II, $LD_{50} > 200$ mg/kg, rabbits tested (EPA, 1986e)	IV, rabbits tested (EPA, 1986e)	IV, rabbits tested (EPA, 1986e)	NA

Table 3-3 (continued)

Dermal toxicology studies of the 14 herbicides and additives

	Acute Dermal	Primary Dermal	Primary Eye	Subchronic Dermal
Triclopyr	III, $LD_{50} \geq 2,000$ mg/kg (ODT), no mortalities, rabbits tested (EPA, 1986f)	IV, rabbits tested (EPA, 1986f)	II, rabbits tested (EPA, 1986f)	NA

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[a] Environmental Protection Agency Labeling Guidelines for Pesticides Applied to Skin or Eyes

I. Irreversible corneal opacity at 7 days; corrosive to skin.
II. Corneal opacity reversible within 7 days; severe skin irritation at 72 hours.
III. No corneal opacity; moderate skin irritation at 72 hours.
IV. No irritation to the eyes; mild or slight skin irritation at 72 hours.

[b] Not available.

severely irritating to the rabbit eye in a primary eye irritation study (21.1 percent a.i.) (EPA, 1986b). Amine and ester formulations of 2,4-D caused local skin inflammation in rabbits undergoing a 21-day dermal test (USDA, 1984).

A 2-year dog feeding study with dose levels of 2,4-D ranging from 0 to 500 ppm (0 to 12.5 mg/kg) established a systemic NOEL of 12.5 mg/kg/day, the highest dose tested (EPA, 1986b). A systemic NOEL of 1,250 ppm (62.5 mg/kg/day) was established, based on a 2-year rat feeding study (EPA, 1986b).

Results from the first year of a chronic feeding study on rats have been reviewed by EPA (1986b). Based on kidney effects reported in the study, a NOEL of 1 mg/kg/day was established; the lowest effect level was 5 mg/kg/day. Using a hundredfold safety factor, EPA has established a provisional acceptable daily intake (PADI) level of 0.01 mg/kg/day (EPA, 1985c).

Fetotoxic and maternal toxic NOEL's of 5 mg/kg/day are based on a one-generation reproduction study with rats exposed to 2,4-D acid at 5, 20, and 80 mg/kg/day. Decreased maternal body weight[1] and reduced pup weight were observed at 20 mg/kg/day (EPA, 1986a). No effects on fertility were seen. Delayed bone ossification in fetuses at 75 mg/kg was observed in teratology studies (EPA, 1986b). No teratogenic effects were observed in the offspring of rats given doses of up to 750 mg/kg 2,4-D (EPA, 1986a). Other studies have reported teratogenic effects including subcutaneous edema, lumbar ribs, wavy ribs, abdominal hemorrhage, enlarged fetal brain cavity, and other anomalies (USDA, 1984).

2,4-D Contaminants

In the case of 2,4-D, special attention must be paid to two contaminants, one of which is also a metabolic product in microogranisms. The issue arises not because of data indicating hazard but because of allegations based on incorrect evaluation of the data.

In the manufacture of 2,4-D, 2-4-dichlorophenol (2,4-DCP) is an intermediate, a minute fraction of which may remain in the final product. It is also an environmental metabolite of 2,4-D. Because of its relatively low toxicity (the LD_{50} is approximately 1,300 mg/kg), 2,4-DCP has not been judged sufficiently toxic to be eliminated from 2,4-D formulations.

The effects of 2,4-DCP on human health have not been well studied. Boutwell and Bosch (1959) examined the carcinogenicity of 2,4-DCP and found it to be a weak tumor promoter. It was also found to inhibit oxidative phosphorylation in rat liver and brain mitochondria (Mitsuda et al., 1963). Somani and Khalique (1982) found that after intravenous administration of 2,4-DCP in rats, the chemical was rapidly metabolized to glucuronide and other conjugates and was eliminated from the body. They showed that half-lives in the kidney and liver are longer than in other tissues, indicating that the liver is a major organ for metabolism, and that the higher levels in the kidneys correlate with that being the route of elimination. Seyler et al. (1984) performed some preliminary reproductive screening procedures and found that 2,4-DCP did not depress sperm penetration of ova and sperm motility in vitro when compared with controls. A 2,4-DCP teratology study recently reviewed by EPA found a NOEL of 350 mg/kg/day; the lowest effect level was found to be 750 mg/kg/day with the effect being delayed ossification (EPA, 1985f). In conclusion, 2,4-DCP appears to be less toxic than the parent herbicide 2,4-D. 2,4-DCP is the immediate microbial breakdown product of 2,4-D and is in turn further oxidized by the same organisms. The rate function for each of the steps in this long series of oxidations is higher than the preceding step.

Breakdown thus becomes easier with each step. The products are mostly not liberated but reamin captive in the microorganims.

2,4-DCP is so volatile that if it were to escape it would immediately dissipate. It also has an exceedingly low olfactory threshold; extremely small amounts are detectable by smell. Because of these factors, only applicators or others working directly with the material before it is applied have any significant opportunity for contact.

The eight manufacturers of 2,4-D in the United States have subjected their products to analysis for 2,4-DCP. Total chlorophenols, of which 2,4-DCP is predominant, were about 0.3 percent in the most contaminated sample. Therefore, at worst, such immediate contact is something less than 0.3 percent of the corresponding exposure to 2,4-D. Many contained no detectable chlorophenols. Other chlorophenols include 2,6-DCP and the 2-chloro- and 4-chlorophenols, all of which are minor contributors (Warren, 1983).

Environmental exposures will not correspond to the amount of 2,4-D applied, either as a fixed fraction of impurity or as a fraction of applied and degraded 2,4-D. As an impurity, 2,4-DCP has a high vapor pressure, so it evaporates and disappears quickly. As a metabolite of soil organisms, 2,4-DCP is almost entirely entrained in those organisms, although at high levels of 2,4-D in water some DCP can be found. Environmental exposure to 2,4-DCP is so low that it cannot be measured.

The other impurity of concern in 2,4-D formulations is 2,7-dichloro dibenzo-p-dioxin (DCDD), which differs only slightly in structure from the well-known 2,3,7,8 TCDD, but differs by about a millionfold in toxicity. Two concerns of biological danger have been expressed: DCDD is alleged to be a teratogen and is alleged to be carcinogenic.

DCDD has been found in 3 of 30 samples of U.S.-produced 2,4-D, along with traces of other relatively nontoxic chlorodioxins with three and four chlorines. The concentrations in the three positive samples ranged from 25 to 60 ppb. Tf the maximum expected human dose of 2,4-D is 0.1 mg/kg, and for convenience all 2,4-D is assumed to contain 100 ppb of DCDD, the dose of DCDD to the exposed human would be 0.00000001 mg/kg.

The toxicologic studies from which these concerns arise are reported by Khera and Ruddick (1973), who discussed fetotoxic effects of DCDD, and the National Cancer Institute (1979), which conducted carcinogenesis studies in two species. Khera and Ruddick fed DCDD at dosages of 1 and 2 mg/kg daily to determine whether DCDD could cause birth defects. The observed effect at 1 mg/kg was a modest degeneration of heart muscle fibers and some fluid accumulation around the heart in a few of the animals. A somewhat greater number of animals were affected at 2 mg/kg. Both effects are in the category of general fetal toxicity. No teratogenic effect was found.

The National Cancer Institute (1979) work was carried out by feeding DCDD as 0.5 and 1 percent of the total diet for 2 years. The data indicated a "suggested" carcinogenic effect in male mice that was not strong enough to support a conclusion that DCDD is a carcinogen. Male mice and rats of both sexes did not significantly respond.

The conclusion, therefore, is that neither 2,4-DCP nor 2,7-DCDD, at maximum occupational or environmental exposures to 2,4-D, represents a human hazard.

2,4-DP

2,4-DP is classified as slightly toxic based on the acute oral LD_{50} of 532 mg/kg for rats (EPA, 1984a). Studies reviewed by EPA (1984a) included a subchronic 90-day rat feeding study and a 2-year feeding/oncogenic rat study that both established a NOEL of 5 mg/kg. At 15 mg/kg/day, decreases in urinary specific gravity and/or protein in males were observed. At 25 mg/kg/day, packed cell volume and blood sodium levels were decreased, and kidney and liver weights were increased. A systemic NOEL of 100 mg/kg/day based on effects to the liver was established for an 18-month mouse feeding study. Two-year feeding studies with rats determined a systemic NOEL of 50 mg/kg/day. Effects observed at the LEL of 150 mg/kg/day included decreased weight gain, decreased hematocrit and red blood cells, chronic prostatitis, and kidney degeneration (EPA, 1984a). Mild skin irritation was observed on 2,4-DP-treated rabbits, with complete recovery in 24 hours (EPA, 1984a). Weedone, a formulation of 2,4-DP, caused no irritation to rabbit skin in a primary dermal test (EPA, 1984a). Slight eye irritation was observed when rabbits underwent a primary eye irritation study (EPA, 1984a).

2,4-DP appears to cause fetotoxic, maternal toxic, and teratogenic effects in laboratory animals. A fetotoxic NOEL of 6.25 mg/kg/day was reported for a three-generation rat reproduction study, with increased mortality of pups occurring at 25 mg/kg/day (EPA, 1984a). In this same study, increased pup mortality during lactation period, reduced maternal body weight, and increased number of smaller litters occurred at the 100 mg/kg dose level. A rabbit teratology study determined fetotoxic and maternal NOEL's of 25 mg/kg and a teratogenic NOEL of less than 25 mg/kg, which was the lowest dose tested (EPA, 1984a). Teratogenic effects characterized by displaced kidneys, omphalocele (navel hernia), and distorted ribs occurred at 25 mg/kg in rabbits (EPA, 1984a). Fetotoxic effects, such as reduced fetal weight and reduced crown-rump distance, were reported at a dose level of 100 mg/kg/day in rabbits (EPA, 1984a). Maternal toxic effects, such as unsteadiness in gait, reduced food intake, and mortality, also were observed at the rabbit dose level of 100 mg/kg/day (EPA, 1984a).

Dicamba

Based on an acute oral LD_{50} of 757 mg/kg in the rat, dicamba is classified as slightly toxic (USDA, 1984). Available data indicate that technical dicamba is a mild eye irritant, but it has a low primary skin irritation toxicity (EPA, 1983b, 1986g). Dicamba, however, can cause a moderate dermal sensitization reaction (EPA, 1986g). A 90-day subchronic feeding study resulted in a NOEL of 25 mg/kg/day as a consequence of slight liver cell alterations (EPA, 1984b). A 15-week rat feeding study in which male Wistar rats (20/dose) were fed diets containing technical dicamba at 0, 31.6, 100, 316, 1,000, or 3,162 ppm (0, 1.6, 5, 15.8, 50, or 158 mg/kg/day) showed liver-to-body weight ratio increases at the 2 highest doses (EPA, 1987e). The NOEL for this study was determined to be 15.8 mg/kg/day. A number of other subchronic rat studies did not reveal adverse effects at any of the doses tested (EPA, 1986g). For example, a 2-year rat study resulted in a systemic NOEL of greater than 125 mg/kg body weight, the highest dose tested.

Fetotoxic and maternal toxic effects have been observed in laboratory animals exposed to dicamba. A rabbit teratology study resulted in setting a maternal and a fetotoxic NOEL of 3.0 mg/kg due to reduced body weights and increased post implantation loss of fetuses, and slightly lower net weight gain in pregnant females (EPA, 1986g). Dicamba was not found to be teratogenic in any of the reported teratology studies (EPA, 1986g). In a three-generation reproduction study, no reproductive effects were observed at 25 mg/kg/day (HDT) (EPA, 1986g).

Fosamine

Using the acute oral LD_{50} of 24,400 mg/kg in the rat for the formulated product (43 percent a.i.) (EPA, 1987a), fosamine is classified as very slightly toxic. Acute and subchronic effects caused by ingestion of fosamine in laboratory animals include weight loss, diarrhea, salivation, prostation, and irregular respiration (USDA, 1984). Acute inhalation exposure for 4 hours caused nasal and ocular discharge, corneal opacity, lung noise, weight loss, and weakness in rats (USDA, 1984). A systemic NOEL of 25 mg/kg/day was reported from a 6-month dog feeding study, with increased stomach weight being the only toxic effect noted (Schneider and Kaplan, 1983, as cited in USDA, 1984). A systemic NOEL of 50 mg/kg/day (HDT) was established, based on a 90-day rat feeding study (DuPont, 1983a). An acute dermal study showed that a fosamine formulation (69 percent reaction products and 43 percent a.i.) is a skin irritant (EPA, 1987a). No irritation was observed when the fosamine formulation was tested in primary and subchronic dermal studies using guinea pigs and rabbits, respectively (EPA, 1987a).

Although Dupont (1983a) reported no fetotoxic, teratogenic, or reproductive toxic effects in rats in a one-generation reproduction study and a teratology study at the highest doses tested of 250 to 500 mg/kg/day and 500 mg/kg/day, respectively, CDFA (1986) reported hydronephrosis at the highest dose tested, resulting in a teratogenic NOEL of 50 mg/kg/day.

Glyphosate

Based on the acute oral LD_{50} of 4,320 mg/kg in the rat, glyphosate is classified as slightly toxic (table 3-1) (EPA, 1986c). A rat oral LD_{50} of 5,600 mg/kg has been reported by Monsanto (1982a,b, as cited in USDA, 1984), but this has not been reviewed by EPA. A 26-month rat feeding study reported no observable effects at the highest dose tested (EPA, 1986c). Using this study, EPA established a NOEL of greater than 31 mg/kg/day (HDT). A recent 1-year chronic feeding study in dogs reported no compound-related effects at the highest dose of 500 mg/kg/day (EPA, 1987b). In a mouse chronic feeding/oncogenicity study, liver cell damage was observed at the highest dose of 4,500 mg/kg/day (EPA, 1986j). The NOEL for this study was therefore established as 750 mg/kg/day. EPA (1986c) reported that severe erythema (redness) occurred when rabbits' eyes were treated with glyphosate in a primary eye irritation study. A primary dermal study showed that no irritation occurred in rabbits tested (EPA, 1986c). A NOEL of 1,000 mg/kg/day was established in a 21-day dermal test using rabbits (EPA, 1986c).

A three-generation reproduction study of glyphosate in rats established a NOEL of 10 mg/kg/day (EPA, 1986c). This NOEL was based on renal tubular dilation in the kidneys of the pups. No effects on fertility or reproductive parameters were noted. Based on this study, EPA has established an ADI level of 0.1 mg/kg/day (EPA, 1986c). In two rat and rabbit teratology studies, no evidence of teratogenicity was observed (EPA, 1986j). In the rat study, evidence of developmental toxicity in the form of unossified sternebrae was observed in fetuses at 3,500 mg/kg/day (EPA, 1986j). This dose was also toxic to dams as evidenced by weight gain deficits, altered physical appearance, and mortality. The rat fetotoxic and maternal toxic NOEL's were therefore established at 1,000 mg/kg/day for this study.

In the rabbit teratology study, the highest dose (350 mg/kg/day) was toxic to does as evidenced by altered appearance and mortality (EPA, 1986j). No treatment-related fetal effects were observed. The maternal toxic NOEL for this study is 175 mg/kg/day and the fetotoxic NOEL is 350 mg/kg/day (HDT).

A nitrogen derivative of glyphosate, N-nitrosoglyphosate (NNG), occurs as a contaminant of technical glyphosate at a level of 0.1 mg/kg or less (EPA, 1986j). EPA (1986j) has classified NNG as slightly toxic (toxicity category III) and has concluded that because the amount of NNG is less than 1.0 mg/kg, no additional toxicology data are required. Monsanto (1986) has conducted a number of studies on NNG and has concluded that it is not teratogenic, mutagenic, or oncogenic.

Hexazinone

Hexazinone is classified as slightly toxic based on the acute oral LD_{50} of 1,690 mg/kg (EPA, 1986d). The systemic NOEL's based on 2-year mouse and rat feeding studies were established as 30 mg/kg/day (mice) and 10 mg/kg/day (rats) (EPA, 1986d). The toxic effects observed during the mouse study included increased liver size, a localized increase in size and number of liver cells, and localized tissue degeneration at the LEL of 375 mg/kg/day. Effects observed in rats included reduced body weight gain, decreased food consumption, increased leukocyte counts, and excretion of a more alkaline urine (EPA, 1982a). Acute and primary dermal studies revealed that hexazinone caused reversible irritation in rabbits (EPA, 1982a, 1986d). Reversible corneal opacity occurred in rabbits treated with hexazinone in a primary eye irritation study (EPA, 1986d).

In a 90-day rat feeding study, the only effect noted was reduced body weight gain at 250 mg/kg/day (HDT) (EPA, 1982a). Slight liver effects and reduced body weight gain were noted in dogs at 125 mg/kg/day in a 3-month feeding study (EPA, 1982a).

In a three-generation reproduction study, no effects on reproduction or lactation performance were observed in rats at the highest dose (125 mg/kg/day) (EPA, 1982a). However, the average body weight of pups at weaning was slightly lower at 125 mg/kg/day. Thus, the reproductive NOEL was established at 125 mg/kg/day, and the fetotoxic NOEL was established at 50 mg/kg/day.

Hexazinone was not embryotoxic or teratogenic at 150 mg/kg/day (HDT) in a rat teratology study (EPA, 1982a). Likewise, no teratogenic effects were observed in rabbits at 125 mg/kg/day (HDT) in a teratology study (EPA, 1982a).

Imazapyr

Based on an acute oral LD_{50} of greater than 5,000 mg/kg in rats, imazapyr is considered very slightly toxic to mammals (EPA, 1985b). Other imazapyr studies reviewed by EPA (1985b) included an acute dermal toxicity study that reported an LD_{50} of greater than 2,000 mg/kg body weight in rabbits. In primary irritation studies, imazapyr was irritating to the eyes and mildly irritating to the skin of rabbits. A dermal sensitization test was negative in guinea pigs. A 21-day dermal study in rabbits showed no signs of systemic toxicity at 400 mg/kg/day. American Cyanamid (1985a) reported a 13-week rat feeding study that established a NOEL of 500 mg/kg/day (HDT). A maternal toxic NOEL of 300 mg/kg/day, based on salivation at 1,000 mg/kg/day, was established in a rat teratology study (EPA, 1985b). However, no teratogenic or fetotoxic effects were observed in rats at 1,000 mg/kg, the highest dose tested, or in rabbits at 400 mg/kg, the highest dose tested (EPA, 1985b; American Cyanamid, 1985a). Chronic studies in rats and dogs are in progress (American Cyanamid, 1987).

Light Fuel Oil (Diesel Oil and Kerosene)

Using an acute oral LD_{50} of 9.0 ml/kg (7,380 mg/kg)[1], diesel oil is classified as a very slightly toxic compound (Beck et al., 1982). The most marked acute toxic effect observed after the administration of diesel oil to test animals occurred during primary dermal irritation studies (Beck et al., 1982). In these studies, a single exposure of rabbits to diesel oil resulted in a rating of "extremely irritating," based on a score of 6.82 (on a scale of 1 to 10). The irritation may have been caused by additives for internal combustion in diesel oil. Diesel oil was nonirritating in primary eye irritation studies (Beck et al., 1982). A subacute 3-week dermal study of eight rabbits reported an average weight loss of 0.38 kg at the dose level of 4.0 ml/kg (3,280 mg/kg) and an average weight loss of 0.55 kg with a 67-percent mortality rate at the dose level of 8.0 ml/kg (6,560 mg/kg) (Beck et al., 1982). An inhalation teratology study in which rats were exposed to 5.09 or 20.075 ul/kg of diesel fuel on days 6 through 15 of gestation did not result in any significant teratogenic effects (Mecler and Beliles, 1979).

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[1] One ml of diesel oil weighs 820 mg.

Kerosene is classified as very slightly toxic, based on the lowest oral lethal dose of 28,000 mg/kg in rats (HSDB, 1987a). Kerosene and all other hydrocarbons represent an acute ingestion hazard to humans. They can lead to chemical pneumonia and should never be swallowed (HSDB, 1987a). Chemical pneumonitis from hydrocarbons, such as kerosene, is described in Doull et al. (1980) as follows:

> An important toxicologic problem associated with the hydrocarbon solvents is the inadvertent or intentional ingestion of gasoline, kerosene, or paint thinners. Although in most instances the acute toxicity of these compounds is quite low, small amounts may be aspirated into the lungs during ingestion, during attempts to induce vomiting, or while pumping the stomach. The response of the lung to small quantities of hydrocarbon solvents is rapid and severe. Relatively small amounts will spread a thin layer over the large moist surfaces of the lung resulting in pneumonitis, pulmonary edema, and hemorrhage.

Kerosene causes moderate local irritation, central nervous system depression, and sometimes mild lesions in the kidneys, liver, bone marrow, and spleen (Gosselin, 1976, as cited in HSDB, 1987a). In a 28-day dermal toxicity study with rabbits, kerosene was moderately irritating at the 200 and 1,000 mg/kg dose levels and was severely irritating at the 2,000 mg/kg dose level (American Petroleum Institute, 1983a). Treatment-related skin lesions (acanthotic dermatitis, hyperkeratosis, and dermal microabscesses) and liver lesions (acute multifocal necrosis) occurred at the highest dose (2,000 mg/kg/day). Jet fuel A (a type of kerosene) was mildly irritating to the skin and eyes of rabbits in primary skin and eye studies. No reactions were observed for guinea pigs used in the same studies (Beck et al., 1982). Rats exposed to 300 mg/m^3 for 14 to 75 weeks exhibited morphologic changes (such as thickening, congestion, and presence of infiltrates) and cytoenzymatic changes (increased/decreased enzyme activity) in the lungs and kidneys and showed disorders of their acid-base equilibrium (Starek and Kaminski, 1981 and 1982). In a study in which baboons were administered kerosene by various routes, the primate brain appears to be resistant to direct toxic effects of kerosene (Wolfsdorf and Paed, 1976). The authors believe this shows that the lung and liver are able to filter out sufficient amounts of large doses to protect the brain. Jet fuel A was not reported to be teratogenic in a rat inhalation study at the highest dose tested (400 ppm) (Beliles and Mecler, 1982).

Limonene

Based on an acute oral LD_{50} of 5,000 mg/kg in rats, limonene is classified as very slightly toxic (HSDB, 1987b). Limonene is used as a flavoring in many foods and may be found in amounts of up to 2,300 mg/kg, as in chewing gum (Furia and Bellanca, 1975). The acute inhalation LD_{50} is greater than 5 mg/l in rats (JLB International Chemical, Inc., 1983). Limonene caused moderate skin irritation in rabbits administered 500 mg/24 hours dermally (HSDB, 1987b). Rats given oral doses of 227 to 1,385 mg/kg/day showed a slight decrease in body weight and little or no change in water and food consumption (Tsuji et al., 1975, as cited in HSDB, 1987b). In this study, no histopathological changes were noted except for granular casts in the kidneys of some males. Oral doses of 400 mg/kg/day for 30 days in rats caused decreased plasma and liver cholesterol, increased enzymes, and altered fatty acids of liver phospholipids (Ariyoshi et al., 1975, as cited in HSDB, 1987b). Dogs administered 1.2 to 3.6 ml/kg/day through inhalation exhibited frequent vomiting and nausea and decreased body weight, blood sugar, and cholesterol (Tsuji et al., 1975, as cited in HSDB, 1987b). In this study, no significant changes were observed in the organs except in the kidneys. In a mouse teratology study, decreased body weight gain and increased abnormal fetal bone formation were caused when females were given 2,363 mg/kg/day during days 7 to 12 of gestation (Kodama et al., 1977, as cited in HSDB, 1987b). Since no studies are available that resulted in a level where no effects were observed, the reproductive/developmental NOEL was set at 227 mg/kg/day, based on the systemic NOEL.

Picloram

With an acute oral LD_{50} of 8,200 mg/kg in rats (EPA, 1984c), picloram is classified as very slightly toxic. A 6-month dog feeding study, during which test animals were exposed to picloram at the dietary levels of 0, 7, 35, and 175 mg/kg/day, resulted in establishing a subchronic NOEL of 7 mg/kg/day (Barna-Lloyd et al., 1982, as cited in Mullison, 1985). Increased liver weights were reported at the lowest effect level of 35 mg/kg/day in males. Other subchronic feeding studies resulted in slight liver effects at 150 mg/kg/day in rats and at 1,000 mg/kg/day in mice (EPA, 1984f). Slight eye and skin irritation was observed in primary eye and primary and acute dermal irritation studies using rabbits (EPA, 1984c).

In a recent 2-year chronic toxicity-oncogenicity study reported by Dow (1987a), rats fed 20 mg/kg/day showed no treatment-related effects. Rats given 60 and 200 mg/kg/day exhibited increased size and altered properties of liver cells. No other chronic feeding studies have been reported; EPA has requested a chronic nonrodent feeding study for picloram (EPA, 1984f).

In a 3-generation rat reproduction study, a NOEL of 50 mg/kg/day was established based on reduced fertility at the highest dose tested of 150 mg/kg/day (EPA, 1988). In a rat teratology study, maternal toxicity was observed at 750 mg/kg and fetal toxicity (delayed bone ossification) was observed at 500 mg/kg (EPA, 1984f). No teratogenic effects were observed, and the NOEL was established as greater than 1,000 mg/kg, the highest dose tested. No dose-related embryotoxic or teratogenic responses were observed in rabbits given doses of picloram of up to 400 mg/kg/day (John-Greene et al., 1985).

Sulfometuron Methyl

Sulfometuron methyl is very slightly toxic, based on an acute oral LD_{50} of greater than 5,000 mg/kg in rats (EPA, 1984c). In acute dermal studies, an LD_{50} of greater than 2,000 mg/kg was reported (EPA, 1984d). Reversible eye and skin irritation was observed in primary eye and primary dermal irritation studies using rabbits (EPA, 1984d). A 90-day rat feeding study established a systemic NOEL of 50 mg/kg/day, based on hematological effects observed at 250 mg/kg/day (EPA, 1984d). A combined 2-year rat feeding and two-generation reproduction study reported by DuPont (1986) established a systemic NOEL of 2.5 mg/kg/day. In this study, hemolytic effects, liver toxicity, and decreased mean absolute body and brain weights, but not the brain-to-body weight ratio, were observed at 250 mg/kg/day. Hemolytic effects and liver toxicity were also observed at 25 mg/kg/day. In a 1-year dog feeding study, a systemic NOEL of 5 mg/kg/day was reported (EPA, 1984d). Effects observed in dogs included decreased number of red blood cells and increased liver weight at 25 mg/kg/day.

In the two-generation rat reproduction study, a NOEL of 25 mg/kg/day was established, based on reduced maternal food consumption and body weight gains and reduced numbers of offspring (DuPont, 1986). A one-generation rat reproduction study resulted in the establishment of a reproductive NOEL of greater than 250 mg/kg/day (HDT) (EPA, 1984d). A rat teratology feeding study reported reduced body weight gain at 250 mg/kg/day and maternal and fetal toxic NOEL's of 50 mg/kg/day (EPA, 1984d). No teratogenic effects were observed at 250 mg/kg/day, the highest dose tested. A rabbit teratology study was negative for teratogenic, maternal, and fetal toxic effects at 300 mg/kg, the HDT (EPA, 1984d).

Tebuthiuron

No EPA-validated studies exist for assessing acute dermal or acute oral toxicity of tebuthiuron (EPA, 1987c). Of the many studies reported by EPA, the lowest acute oral LD_{50} was 644 mg/kg in rats. Based on this study, tebuthiuron is classified as slightly toxic (EPA, 1986e). Elanco (1989) reports a lower oral LD_{50} in rats, in the range of 388 to 478 mg/kg. A systemic NOEL of 83.1 mg/kg/day (EPA, 1984e) was established from a 119-day mouse feeding study. In a recent registration document from EPA (1987c), a systemic NOEL of 25.0 mg/kg/day was found in a 1-year dog feeding study. A more conservative systemic NOEL of 12.5 mg/kg/day was established for a 3-month dog feeding study, based on increased thyroid-to-body weight values and increased blood enzyme levels (EPA, 1986e). Toxic effects in other subchronic studies included growth suppression and pancreatic lesions at 125 mg/kg/day in rats, and body weight depression at 37.5 mg/kg/day in cattle (EPA, 1986e). Tebuthiuron caused no eye or skin irritation in rabbits during primary eye and primary dermal studies (EPA, 1986e). Tebuthiuron, however, was skin irritating in an acute dermal study using rabbits (EPA, 1986e).

A three-generation reproduction study with rats reported a reproductive NOEL of less than or equal to 20 mg/kg/day (LDT), based on the decreased body weight of weanling pups (EPA, 1986e). In a two-generation reproduction study with rats, EPA reported a NOEL of 5.0 mg/kg/day, based on a low rate of maternal body weight gain (EPA,1987f).

The only study of teratogenicity supplied to EPA was found invalid. Two mammalian teratogenic studies are required to complete reregistration standards for tebuthiuron (EPA, 1987c). The study performed did show that there were no observable teratogenic effects at the highest dose tested (90 mg/kg) (EPA, 1986e).

Triclopyr

With an acute oral LD_{50} ranging from 630 to 729 mg/kg in rats (EPA, 1986f), triclopyr is classified as slightly toxic (table 3-1). A systemic NOEL of 30 mg/kg/day was established, based on a 90-day rat feeding study that resulted in decreased body weight, food consumption, and absolute liver weights (EPA, 1986f). A 2-year feeding/oncogenic study observed no effects on hematology, clinical chemistry, and urinalysis at 30 mg/kg/day (HDT) (EPA, 1986f). In a recent 2-year chronic toxicity-oncogenicity study reported by Dow (1987a), no toxicological effects were observed in rats at 3 mg/kg/day. Male rats fed 12 and 36 mg/kg/day had increased absolute and relative kidney weights. Acute and primary dermal tests revealed that triclopyr was slightly irritating to the skin of rabbits (EPA, 1986f). A primary eye irritation test demonstrated that triclopyr was irritating to rabbit eyes (EPA, 1986f).

A 228-day dog feeding study resulted in a systemic NOEL of less than 5 mg/kg/day, based on decreased weight gain and food consumption (Dow, 1983, as cited in USDA, 1984; EPA, 1986f). A 6-month feeding study with dogs resulted in the establishment of a systemic NOEL of 2.5 mg/kg (HDT) (40 CFR Part 180 50(84):184-85, May 1, 1985). The effects found in the dog studies are not representative of effects expected in humans because dogs have a limited capacity for organic anion transport in the kidney (Dow, 1985). Dogs excrete triclopyr at a slower rate than other laboratory animals or humans. The half-life of triclopyr for urinary excretion in dogs is 96 hours, compared to 1.5 hours in rats and 3.1 hours in monkeys. Dow concluded that toxicity may be increased in dogs because of the greater relative retention time of the compound in the animal's body. Therefore, the use of the NOEL from the dog study (the lowest NOEL found in the literature) in this risk assessment is very conservative and tends to cause an overestimate of expected effects in humans with normal renal function.

In a rat study, teratogenic effects were not observed at 200 mg/kg/day, the highest dose tested (EPA, 1986f). However, the fetotoxic NOEL was reported as 50 mg/kg/day, based on retarded ossification of skull bones. The maternal NOEL was established as less than 50 mg/kg/day, based on reduced body weight gain and food consumption. A three-generation rat reproduction study reported a reproductive NOEL of greater than 30 mg/kg/day (HDT) (EPA, 1986f). No teratogenic effects were observed in two rabbit teratology studies, although one study reported fetotoxic effects at the lowest dose of 10 mg/kg/day (EPA, 1986f). Based on this evidence that the actual reproductive NOEL may be less than 10 mg/kg/day, it was set at 2.5 mg/kg/day for the purposes of this risk assessment, in correlation with the systemic NOEL.

Animal Metabolism and Elimination

The herbicides evaluated in this risk assessment are rapidly excreted when administered to animals. Elimination of 90 percent or more, within 2 hours to 5 days, was reported for most of the 11 herbicides. Table 3-4 displays the elimination rates of the 14 chemicals. In addition to the rapid elimination of the herbicides, tissue retention studies showed low residue concentrations in animal tissues (USDA, 1984).

Based on the high elimination rates and low tissue retention, the herbicides used for Region 8 vegetative management present a very low risk for bioaccumulation. Bioaccumulation analyses were therefore not conducted for this risk assessment.

MUTAGENICITY OF THE 14 HERBICIDES AND ADDITIVES

This subsection presents a review of the available information on the mutagenic hazard of the 14 chemicals. Table 3-5 summarizes the tests on each of the herbicides and light fuel oil for each category of testing recommended by EPA in their guidance documents on mutagenicity (EPA, 1978, 1986j). The source used for summarizing the mutagenicity tests is defined for each pesticide at the bottom of the table. Mutagenic assays that did not fall into any of the categories are not listed in the table. Table 3-5 also presents the relevance of the recommended tests to a determination of human mutagenic potential according to Dr. David Brusick of Litton Bionetics, Inc., author of Principles of Genetic Toxicology (1980).

EPA has adopted the battery of tests scheme in order to assess the potential mutagenic hazard of chemicals. Three groups of tests are used to detect gene mutations, chromosomal aberrations, and primary DNA damage. Tests in each category have their own strengths and weaknesses in determining mutagenicity. This testing scheme is designed such that the strengths of some tests cover areas where other tests are weak. All test results within a group are not expected to be the same (Brusick, 1980). Thus, the determination of the mutagenic potential of a chemical must be based on the weight-of-evidence from the battery of tests, with consideration to each test's ability to predict human mutagenic effects.

Table 3-4

Elimination rates of the 14 herbicides and additives[a] considered for use in Region 8

Chemical	Test Animal	Elimination Rate
2,4-D	Rat Rat	93% within 2 hours (Grissom et al., 1985) 100% within 5 days (Fisher et al., 1985)
2,4-DP	Rat	74% to 82% within 4 days (EPA, 1984a)
Diesel Oil	NA[b]	NA[b]
Dicamba	Rat Mouse	100% within 48 hours (EPA, 1984b) 99% within 4 days (EPA, 1984b)
Fosamine	Rat	99 to 100% within 72 hours (USDA, 1984)
Glyphosate	Rabbit Rat	92% within 5 days (USDA, 1984) 94% within 5 days (FAO/WHO, 1986)
Hexazinone	Rat Rat	93% within 24 hours (USDA, 1984) 94.2 to 100% within 72 hours (USDA, 1984)
Imazapyr	Rat	87% within 24 hours (American Cyanamid, 1985b)
Kerosene	NA	NA[b]
Limonene	NA	NA[b]
Picloram	Dog Unspecified	90% within 48 hours (USDA, 1984) 96% within 24 hours (Nolan et al., as cited in Lavy and Mattice, 1986)
Sulfometuron methyl	NA	NA[b]
Tebuthiuron	NA	NA[b]
Triclopyr	Rat	83% to 91% within an unspecified period (USDA, 1984)

[a] The 9 chemicals for which information is available were excreted rapidly (EPA designation) by the mammals tested.

[b] Not available.

Table 3-5

Mutagenicity testing on the 11 herbicides and light fuel oil

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	2,4-D	2,4-DP	Dicamba	Diesel oil	Fosamine	Glyphosate
Group 1--Tests for detecting gene mutations							
A. Bacteria with and without metabolic activation (includes Ames assay)	+	13(-)[d]	2(-)	4(-)	2(-)	2(-)	7(-)
B. Eukaryotic microorganisms with and without metabolic activation (includes yeast assay)	+	1(-) 4(+)	1(+)				
C. Insects (for example, sex-linked recessive lethal test)	++	1(-) 2(+)					
D. Mammalian somatic cells in culture with and without metabolic activation (includes mouse lymphoma assay)	++				2(-)		
E. Mouse-specific locus test *in vivo*	++						
F. Mammalian germ cells in culture with and without metabolic activation[c]						2(-)	2(-)
Group 2--Tests for detecting chromosomal aberrations							
A. Cytogenetic tests in mammals *in vivo* (includes rat bone marrow cell assay)	++	3(-)			1(+)	1(-)	1(-)
B. Insect tests for heritable chromosomal effects *in vivo*	++						
C. Dominant-lethal effects in rodents, heritable translocation tests in rodents, and *in vitro* cytogenetic assays in mammals	++	3(-) 2(+)				2(+)	1(-)
Group 3--Tests for detecting primary DNA damage							
A. DNA repair in bacteria (including differential killing of DNA repair defective strains and recombination assay) with and without metabolic activation	NA	1(-) 2(+)	1(-) 1(+)				1(-)
B. Unscheduled DNA repair synthesis in mammalian somatic cells in culture, with and without metabolic activation	NA	2(-) 1(+)		2(-)		1(-)	1(-)
C. Mitotic recombination and gene conversion in yeast, with and without metabolic activation	NA	3(-) 1(+)	1(-) 1(+)				
D. Sister-chromatid exchange in mammalian cells in culture, with and without metabolic activation	NA	1(+)					

Table 3-5 (continued)

Mutagenicity testing on the 11 herbicides and light fuel oil

Mutagenicity Test Type[a]	Value in Determining Human Mutagenicity[b]	Hexazinone	Imazapyr	Kerosene	Picloram	Sulfometuron Methyl	Tebuthiuron	Triclopyr
Group 1--Tests for detecting gene mutations								
A. Bacteria with and without metabolic activation (includes Ames assay)	+	2(-)[d]	4(-)	2(-)	6(-) 1(+)	1(-)	4(-)	5(-)
B. Eukaryotic microorganisms with and without metabolic activation (includes yeast assay)	+				2(-)			1(-)
C. Insects (e.g., sex-linked recessive lethal test)	++							
D. Mammalian somatic cells in culture with and without metabolic activation (includes mouse lymphoma assay)	++			2(-)			1(+) 1(-)	
E. Mouse-specific locus test *in vivo*	++							
F. Mammalian germ cells in culture with and without metabolic activation[c]		1(-)	1(-)			1(-)		
Group 2--Tests for detecting chromosomal aberrations								
A. Cytogenetic tests in mammals *in vivo* (includes the rat bone marrow cell assay)	++	1(-)		1(-)	1(-)			1(-)
B. Insect tests for heritable chromosomal effects *in vivo*	++							
C. Dominant-lethal effects in rodents, heritable translocation tests in rodents, and *in vitro* cytogenetic assays in mammals	++	2(+)	2(-)			1(-)	1(-)	1(-) 1(+)
Group 3--Tests for detecting primary DNA damage								
A. DNA repair in bacteria (including differential killing of DNA repair defective strains and recombination assay) with and without metabolic activation	NA							1(-)
B. Unscheduled DNA repair synthesis in mammalian somatic cells in culture, with and without metabolic activation	NA	1(-)	1(-)			1(-)		
C. Mitotic recombination and gene conversion in yeast, with and without metabolic activation	NA							
D. Sister-chromatid exchange in mammalian cells in culture, with and without metabolic activation	NA							

[a] Source: FIFRA, Environmental Protection Agency: Proposed Guidelines for registering pesticides in the U.S. Hazard Evaluation: humans and domestic animals. (EPA, 1978, 1986j)

[b] Value in Determining Human Mutagenicity according to Dr. David Brusick, genetic toxicologist of Litton Bionetics, Inc.: NA = Not Applicable; + = Applicable; ++ = Greater applicability

[c] This test type was not included in the FIFRA mutagenicity guidelines but was added to this table to incorporate results of studies such as Chinese hamster ovary cell tests. The value of this test type would be equal to category 1D.

[d] The numerals represent the number of positive (+) and negative (-) results reported for each category.

Source: EPA tox one-liners and registration standards for individual pesticides, 1982-1987, were used to prepare this table unless otherwise noted; USDA (1984) for 2,4-D and picloram; American Cyanamid (1986, 1985a) for imazapyr; Conaway et al. (1982) for diesel oil and kerosene. For fosamine, hexazinone, and sulfometuron methyl, EPA tox one-liners were used in addition to the following: DuPont (1983a) for fosamine; DuPont (1984a) for hexazinone; and DuPont (1983b) for sulfometuron methyl.

In general, for all three test categories, EPA (1986k) places greater emphasis on assays conducted in germ cells than in somatic cells (for detecting heritable mutations), in vivo rather than in vitro, in eukaryotes rather than prokaryotes, and in mammalian species rather than submammalian species. In vivo mammalian systems are considered to be of greater value because of their similarity to human physiology and metabolism. EPA (1986k) classifies the evidence for potential human germ cell mutagenicity as sufficient, suggestive, or limited, depending on the results of various tests performed. For instance, positive results in even one in vivo mammalian germ cell mutation test are considered sufficient evidence for potential human mutagenicity of a specific chemical.

Types of mutagenicity assays were discussed earlier in this chapter. As stated, the most relevant mutagenic assays usually are in vivo studies and germ cell studies (for example, dominant lethal mouse and heritable translocation mouse assays). A mutated mammalian germ cell if fertilized could pose a serious problem for the developing fetus. The individual (if capable of reproducing) would pass the defective genome to the next generation, thereby establishing heritable genetic sickle cell anemia and cystic fibrosis. Thus, germ cell studies are considered relevant to evaluating the heritable mutagenicity of chemicals. In vitro studies using mammalian cells are of less importance because of the high percentage of false positive findings resulting from interactions between the cultured cells and media conditions. Tests for detecting primary DNA damage (group 3 in table 3-5) are not applicable in determining the human mutagenic potential of a chemical.

The majority of tests reviewed were those indicated as valid by EPA in toxicity test summaries (tox one-liners or EPA science chapters). If these sources were not available, studies of mutagenicity were obtained from USDA pesticide background statements, which reported studies from the open literature. Results reported within the same study for different test species or different test types (for example, inactivated and activated assays) were counted as individual tests. Therefore, a single study reported in EPA tox one-liners may be represented more than once in table 3-5. For instance, one study that reported positive results in the Ames reverse mutation test for bacteria Salmonella spp. and E. coli, both activated and inactivated, would represent four positive results in category 1A. Males and females, as well as different strains of the same species, were counted as one test only, unless different results were reported for each.

For some of the herbicides, mutagenicity tests conducted are insufficient to conclude whether the chemical is mutagenic. In these cases, the results of carcinogenicity tests (table 3-5) were used to estimate mutagenic risk, based on a high correlation between mutagenic and carcinogenic activity reported in several studies (Blackburn et al., 1984; Pogodina et al., 1984; Parodi et al., 1981, 1982, 1983a,b). However, because correlations vary greatly according to the class of chemicals and the type of test used, carcinogenicity should not be viewed as a definitive predictor, but rather as a possible indicator of mutagenicity.

2,4-D

No mutagenicity studies were reported on the most current EPA tox one-liner for 2,4-D (EPA, 1986b). Studies not evaluated by EPA have determined negative, weakly positive, and positive mutagenic responses to 2,4-D exposure for various test systems (USDA, 1984; WHO, 1984). Mutagenic assays with 2,4-D have yielded conflicting results in gene mutation tests with eukaryotic organisms and insects, in chromosomal aberration tests with mammals, or mammalian cells, and in primary DNA damage tests in prokaryotic, eukaryotic, and mammalian organisms (USDA, 1984). Conflicting results were reported for many of the tests (USDA, 1984). Tests of 2,4-D for gene mutation in bacteria were all negative (USDA, 1984). Mutagenic and toxic effects in yeast were dependent on low pH levels. Although toxicity to bacteria was pH dependent, mutagenicity was not (USDA, 1984). Newton and Dost (1981) concluded that 2,4-D may be a weak mutagen but that it is "without significance as an environmental mutagenic hazard." EPA has requested additional data to evaluate the mutagenic potential of 2,4-D in mammalian test systems. Although the mutagenicity of 2,4-D is uncertain, 2,4-D is evaluated as if it were mutagenic for this risk assessment.

2,4-DP

2,4-DP was nonmutagenic when tested in two microbial assays, both activated and nonactivated (EPA, 1984a). However, positive results were reported in a nonactivated reverse gene mutation assay with yeast reviewed by EPA (1984a). EPA also reported a bacterial assay that was positive for unscheduled DNA synthesis with metabolic activation, but it was negative without activation. Positive results were reported in yeast for mitotic gene conversion, while negative results were reported for mitotic recombination (EPA, 1984a). Based on the inconsistent genotoxic responses and the positive oncogenic effects observed in a chronic oncogenic feeding study of rats, 2,4-DP is evaluated as if it were mutagenic for this risk analysis.

Dicamba

Bacterial studies with dicamba reported negative results for gene mutation, with and without metabolic activation (EPA, 1986g). In addition, EPA (1986g) reported negative results for unscheduled DNA synthesis with and without activation. EPA (1986g) reviewed five other mutagenicity tests that were judged invalid or unacceptable. In studies reviewed by USDA (1984), dicamba was nonmutagenic in eight of ten tests. Five bacterial point mutation assays and three DNA damage assays were negative for mutagenicity, while two bacterial tests for DNA damage were positive. Based on the available evidence, dicamba is assumed to be nonmutagenic for this risk assessment.

Fosamine

In studies reviewed by EPA (1987a), fosamine caused chromosome aberrations in activated and nonactivated *in vitro* cytogenetic assays of Chinese Hamster ovary cells, but it was negative in a rat cytogenetic *in vivo* assay and a rat DNA damage/repair assay. Other studies reviewed in USDA (1984) reported that fosamine was nonmutagenic when tested with and without metabolic activation in bacterial assay systems and a point mutation assay with mammalian germ cells in vitro. Fosamine is considered nonmutagenic for this risk assessment.

Glyphosate

Glyphosate was nonmutagenic in bacterial assays for gene mutation and primary DNA damage, and it also was nonmutagenic in mammalian assay systems both in vitro and in vivo (EPA, 1986c,j). There is no evidence to indicate that it is mutagenic, so it is considered nonmutagenic for this risk assessment.

Hexazinone

Hexazinone was nonmutagenic in Ames assays, in an in vitro mammalian point mutation assay, in an assay of unscheduled DNA repair synthesis in mammalian somatic cells, and an in vivo mammalian cytogenetic assay (EPA, 1986d; USDA, 1984). Hexazinone induced chromosome damage in an in vitro cytogenetic assay with Chinese hamster ovary cells both with and without metabolic activation (EPA, 1986d). This effect was observed only at very high levels and could be caused as a secondary effect of an (unevaluated) metabolic imbalance, such as high ionic concentrations or pH. Based on these results, hexazinone is considered nonmutagenic to humans for this risk analysis.

Imazapyr

Imazapyr was nonmutagenic in the Ames bacterial assays (with and without metabolic activation), the dominant lethal mouse assay, a Chinese hamster ovary in vitro cytogenetic assay, an unscheduled DNA repair synthesis test, and the Chinese hamster ovary cell HGPRT assay (gene mutation mammalian germ cell test) (American Cyanamid, 1985a, 1986). Based on these results, imazapyr is determined to be nonmutagenic for this risk assessment.

Light Fuel Oil (Diesel Oil and Kerosene)

Diesel oil was nonmutagenic when tested with and without metabolic activation in the Ames assay and the mouse lymphoma assay. However, it was found to be clastogenic (causing chromosomal breaks) in rat bone marrow cells (Conaway et al., 1982). Kerosene was nonmutagenic when tested with and without metabolic activation in the Ames assay, the mouse lymphoma assay, and the rat bone marrow cell assay (Conaway et. al., 1982). However, because diesel oil and kerosene contain polycyclic aromatic hydrocarbons (PAH's) and other constituents that are known or suspected mutagens, they are considered to be mutagens for this risk assessment.

Limonene

No mutagenicity studies of limonene have been reported in the literature or by EPA. Limonene is considered a "Generally Regarded As Safe" (GRAS) chemical by the Food and Drug Administration (Furia and Bellanca, 1975). Limonene is used as a food flavoring agent and can be found in baked goods, gelatin and puddings, and chewing gum. This commonly used chemical has never been suspected of being mutagenic, and, as a result, has never been tested. Thus, limonene is considered to be nonmutagenic for this risk assessment.

Picloram

Picloram was nonmutagenic in bacteria and eukaryotic microorganism assay systems and in the rat *in vivo* cytogenetic assay (USDA, 1984; EPA, 1984c). Picloram was mutagenic in one bacteria assay on a previously untried system using *Streptomyces* spp. (USDA, 1984), which has not been validated for use in the standard battery of tests for mutagenicity. EPA (1984f) determined that another study that reported positive results in human lymphocytes was insensitive and incapable of being used to determine mutagenicity. EPA has requested additional picloram mutagenicity studies. There is no evidence that picloram presents a mutagenic risk to humans. It is considered nonmutagenic in this risk analysis.

Sulfometuron Methyl

Sulfometuron methyl was nonmutagenic when tested in an activated *Salmonella* assay (bacteria gene mutation test) and a Chinese hamster ovary cell assay (mammalian germ cell test) (EPA, 1984c). DuPont (1986) also reported negative results for *in vitro* cytogenetic and unscheduled DNA synthesis assays in mammals. Based on these results, sulfometuron methyl is considered nonmutagenic for this risk assessment.

Tebuthiuron

Tebuthiuron was nonmutagenic when tested with and without metabolic activation in bacterial assay systems, in a dominant lethal rat assay, and in an activated mouse lymphoma cell assay. It was mildly mutagenic in a mammalian somatic test cell without metabolic activation. Based on the battery of tests performed, tebuthiuron is assumed to be nonmutagenic (EPA, 1986e).

Triclopyr

Except for a dominant lethal rat assay in which weakly positive results were observed, triclopyr was nonmutagenic in various test systems, including bacteria and yeast assays, a dominant lethal mouse assay, cytogenetic mammalian assay *in vivo*, and a bacteria recombination assay (EPA, 1986f). Therefore, triclopyr is not considered a potential human mutagen in this risk assessment.

CARCINOGENICITY OF THE 14 HERBICIDES AND ADDITIVES

The following discussion summarizes the results of cancer tests and other chronic tests that have been used to determine whether any of the 14 herbicides and additives being considered for use in Region 8 are carcinogenic. Table 3-6 presents a summary listing of the results of the chronic studies.

Table 3-6

Summary of mutagenicity and oncogenicity of pesticides

Chemical	Mutagenicity	Oncogenicity
Herbicides		
2,4-D	Mutagenic in 13/40 assays (USDA, 1984)	Oncogenic in 1/4 studies (EPA, 1984e; Hazelton Laboratories, 1986; EPA, 1986L); scientific uncertainty (Rueber, 1979, as cited in BLM, 1985)
2,4-DP	Mutagenic in 3/7 assays (EPA, 1984a)	Oncogenic in 1/2 studies (EPA, 1984a)
Dicamba	Mutagenic in 0/6 assays (EPA, 1986g)	Oncogenic in 0/3 studies (EPA, 1986i,L)
Fosamine	Mutagenic in 2/8 assays (EPA, 1987b; DuPont, 1983a)	No chronic studies available (EPA, 1987a; USDA, 1984); oncogenic in 0/2 subchronic studies (USDA, 1984)
Glyphosate	Mutagenic in 0/13 assays (EPA, 1986c)	Possibility of weak oncogenic effect in 1/2 studies (EPA, 1985i,c); scientific uncertainty (EPA, 1986L)
Hexazinone	Mutagenic in 2/7 (EPA, 1986d; DuPont, 1984)	Oncogenic in 0/2 test species (EPA, 1986d)
Imazapyr	Mutagenic in 0/6 assays (American Cyanamid, 1985a)	No oncogenic effects observed during the first 12 months of a 2-year rat study (Biodynamics Inc., undated)
Light fuel oil	Diesel oil--mutagenic in 1/5 assays (Conaway et al., 1982) Kerosene--mutagenic in 0/5 assays (Conaway et al., 1982)	Contains aromatic compounds reported to be carcinogenic
Limonene	No mutagenicity studies reported	No oncogenicity studies reported

Table 3-6 (continued)

Summary of mutagenicity and oncogenicity of pesticides

Chemical	Mutagenicity	Oncogenicity
Picloram	Mutagenic in 1/10 assays (USDA, 1984; EPA, 1984f)	Oncogenic in 1/3 studies (EPA, 1984c; Dow, 1987a)
Sulfometuron methyl	Mutagenic in 0/4 assays (EPA, 1984d; DuPont, 1983b)	Oncogenic in 0/2 studies (DuPont, 1986)
Tebuthiuron	Mutagenic in 1/7 assays (EPA, 1986e)	Oncogenic in 0/2 studies (EPA, 1986e)
Triclopyr	Mutagenic in 1/10 bacterial and cytogenetic assays (EPA, 1986f)	Oncogenic in 0/3 studies (USDA, 1984)

The next subsection on cancer potency summarizes the results of the analysis of tumor data on the four herbicides--2,4-D, 2,4-DP, glyphosate, and picloram--that have tested positive in at least one cancer study or have uncertainty regarding carcinogenicity.

2,4-D

A number of studies have assessed the carcinogenicity of 2,4-D, and thus far, there are no conclusive data demonstrating that 2,4-D is carcinogenic (International Agency for Research on Cancer, 1977; Mullison, 1981; State of Minnesota, 1978, all as cited in USDA, 1984). However, there is also general agreement that none of these studies was adequate (EPA, 1982a; International Agency for Research on Cancer, 1977, as cited in USDA, 1984; WHO, 1984). At least one scientist, Dr. M. Rueber, disputes the conclusion that a carcinogenic effect of 2,4-D has not been shown (Rueber, 1979, as cited in BLM, 1985). EPA has recently reviewed a long-term study on the oncogenic potential of 2,4-D and classified it into Category D, generally used for agents with inadequate human and animal evidence of carcinogenicity or for which no data are available. The current ADI Tracking Report (EPA, 1988b) states that there is evidence of oncogenicity in male rats.

At 106 weeks, a preliminary pathology report from a recent mouse study found that 2,4-D was not oncogenic at dosages of 1, 15, and 45 mg/kg/day (Hazelton Laboratories, 1986).

The link between human exposure to phenoxyacid herbicides and cancer has been examined in several epidemiology studies. In the mid- and late-1970's, Hardell and colleagues (Hardell and Sandstrom, 1979; Eriksson et al., 1981; Hardell et al., 1981) conducted a series of case-control studies in rural Sweden. These studies found a significant increase of five- to sixfold in the relative risk of soft-tissue carcinomas, Hodgkin's disease, and non-Hodgkin's lymphoma (NHL) among farmers using various herbicides. However, because of selection and observation biases and uncontrolled confounding variables, the validity of the studies' results (Colton, 1986) have been questioned. In addition, cohort studies of Swedish agricultural and forestry workers by Wiklund and Holm (1986a,b) do not support the results of Hardell and colleagues.

Recently, Hoar et al. (1986) completed a case control epidemiologic study in Kansas, in which they examined the risk of lymphoma and soft-tissue sarcoma (STS) in men from agricultural herbicide exposure. The study found no association between exposure and STS or Hodgkin's disease. A significant association for NHL and phenoxyacetic acid herbicide exposure, singling out 2,4-dichlorophenoxyacetic acid exposure, was reported. In addition, individuals exposed to herbicides for more than 20 days per year had a sixfold increase in NHL. This study, however, suffers from the same inherent limitations as other case-control studies, mainly that it relies on the subject's and the next of kin's recall of exposure status. If recall is faulty, misclassification occurs. Assessing exposure-disease relationships in these types of epidemiological studies is especially difficult (Thomas, 1986). For example, common exposures to other carcinogenic agents or other factors may result in disease but be undiscovered in the interview and confound the results. Thus, uncontrolled confounding factors in observational epidemiological studies can be particularly troublesome in interpreting the results. The apparent dose-response relationship observed in the Hoar et al. (1986) study for NHL is of public health concern and needs further examination.

A recent review of the Hoar et al. (1986) study conducted for EPA by Brian MacMahon, M.D., Ph.D., of the Harvard School of Public Health, concluded as follows:

> In my opinion the weight of evidence does not support the conclusion that there is an association between exposure to 2,4-D and NHL. It is axiomatic that, except when relative risks are very high--and sometimes even then--no single study will establish an association between an exposure and an outcome. The acceptance of an association depends on a number of studies showing consistent results across populations and across different epidemiologic methods. The study of Hoar et al. is a strong study--strong enough on its own to establish a hypothesis of relationship of exposure to 2,4-D with some small proportion of cases of NHL--a hypothesis that clearly deserves attempts at refutation or support in other populations. When one attempts to place the results of this study among the results of those published previously, the picture becomes very confusing--much more so than if Hoar et al. had been the only study published. Taken as a whole, I

> believe that the weight of evidence indicates that an association between 2,4-D and NHL remains a hypothesis that is still to be tested. I am unwilling to speculate as to whether 2,4-D causes NHL (or some cases of NHL) until the evidence is clear that there is an association between them.

Other recent case-control studies of phenoxy herbicides have been reviewed by the Canadian Centre for Toxicology (1987). A study conducted in western Washington State reported no overall increased risk associated with past occupational exposure to phenoxy herbicides for STS or NHL (Woods et al., 1987). There was an elevated risk of NHL for men who had been farmers, forestry herbicide applicators, and those potentially exposed to phenoxy herbicides for 15 years or more during the period prior to 15 years before cancer diagnosis. However, exposure to 2,4-D was not singled out.

Another study reviewed by the Canadian Centre for Toxicology (1987) is being conducted by the National Cancer Institute in Iowa and Minnesota. Preliminary results indicate no overall increased risk for NHL associated with living or working on a farm, and a slightly elevated (but not significant) risk in persons using 2,4-D (Cantor and Blair, 1986). The investigators have decided to recontact subjects to gather more information on the number of days per year of pesticide use.

Two recent case-control studies conducted in New Zealand were negative for soft-tissue carcinoma (Smith et al., 1984) and NHL (Pearce et al., 1986) in association with phenoxy herbicide exposure.

In a recent cohort study of forestry workers in Ontario, no evidence of increased mortality risk or cancer risk was observed after 15 or more years of employment associated with phenoxy herbicide use (Green, 1986). The forestry workers had been employed by Ontario Hydro during the period 1950 through 1982.

Following the review of 2,4-D epidemiology studies, the Canadian Centre for Toxicology (1987) concluded that there is limited evidence of carcinogenicity in man from exposure to phenoxy herbicides, and there is inadequate evidence to classify 2,4-D as a carcinogen.

Now under way are at least two more studies that should be helpful in assessing risks to humans from the use of 2,4-D and other phenoxy herbicides (Colton, 1986). Because of the uncertainty about the carcinogenicity of 2,4-D, a cancer risk analysis will be conducted for 2,4-D in this risk assessment.

2,4-DP

Available evidence indicates that 2,4-DP is carcinogenic in rats (EPA, 1982b). A 2-year feeding study with rats showed tumor formation at doses as low as 25 mg/kg/day (EPA, 1984a). At all doses tested (25, 50, or 150 mg/kg), malignant tumors were induced in test animals. Another study using mice as the test species showed no oncogenic effects at the highest dose tested (300 mg/kg/day) (EPA, 1984a). 2,4-DP is assumed to be a human carcinogen for the purposes of this analysis, and a risk assessment is presented in section 5. 2,4-DP's cancer potency is discussed in the next subsection.

Dicamba

Available evidence does not indicate that dicamba is carcinogenic. A 2-year rat feeding/oncogenic study resulted in the absence of any toxic or oncogenic effects of dicamba at 25 mg/kg/day (HDT) (EPA, 1986g). No oncogenic effects were reported in a 2-year dog feeding study; the only effect seen was decreased body weight (EPA, 1986g). Although the dog study was not conducted as a cancer study, it does provide the results of pathologic analyses after long-term exposure. EPA has requested additional cancer studies for dicamba because the available studies are not considered adequate for defining the oncogenic potential of dicamba based on EPA guidelines under FIFRA (EPA, 1985a).

A recent 2-year rat study accepted by EPA (1986i) showed no oncogenic or systemic effects at the highest dose tested (125 mg/kg/day). For this risk assessment, dicamba is considered nononcogenic.

Fosamine

Very limited data are available regarding the carcinogenic potential of fosamine. In a 6-month dog feeding study, oncogenic effects were not noted at the highest dose tested of 125/187.5/250 mg/kg/day (125 mg/kg/day for 1 week, 187.5 mg/kg/day for 2 weeks, and 250 mg/kg/day for the remainder) (Schneider and Kaplan, 1983, as cited in USDA, 1984). In a 90-day rat feeding study, no oncogenic effects were apparent in rats fed 250/500 mg/kg/day (Schneider and Kaplan, 1983, as cited in USDA, 1984). However, these two studies were not conducted specifically to determine the potential for fosamine to cause cancer. No 2-year chronic feeding/oncogenicity studies have been reported for fosamine. Therefore, there are insufficient data to determine the cancer risk for fosamine in this risk assessment.

Glyphosate

A 26-month rat feeding study found no oncogenic effects at doses up to 31 mg/kg day (EPA, 1986g). However, this study has been downgraded to supplementary by EPA because the maximum tolerated dose (MTD) was not reached at the high dose. Benign kidney tumors (renal tubular adenomas; 3/50) were found at a highest dose level (4,500 mg/kg/day), as well as in the control group (1/50) in a 2-year mouse feeding study. However, the findings were equivocal (EPA, 1986j). The EPA Science Advisory Panel (SAP) has reviewed all relevant data and proposed that glyphosate be classified as a "class D oncogen" or having "inadequate animal evidence of oncogenicity" and that the study be repeated to clarify these findings (EPA, 1986j).

Following a review of the available carcinogenicity studies, the Food and Agriculture Organization and World Health Organization (1986) jointly concluded that there is no evidence that glyphosate is carcinogenic.

EPA, however, is requiring that the mouse study be repeated with more animals in each test group to increase the statistical significance of the study. In view of the uncertainty about the carcinogenicity of glyphosate, a cancer risk analysis will be conducted in this risk assessment.

Hexazinone

Available evidence does not indicate that hexazinone is carcinogenic. In 2-year mouse and rat feeding studies, no oncogenic effects of hexazinone were observed at any of the doses tested (10, 50, and 125 mg/kg/day in rats, and at the testing levels of 30, 375, and 1,500 mg/kg/day in mice) (USDA, 1984). Hexazinone is considered nononcogenic for this risk assessment.

Imazapyr

No evidence of carcinogenicity was observed within the first 12 months of a chronic feeding/oncogenicity study in rats fed 500 mg/kg/day, the highest dose tested (Biodynamics, Inc., undated). An 18-month mouse oncogenicity study is currently in progress (American Cyanamid, 1987). Further study results must be obtained before the carcinogenic potential of imazapyr can be determined.

Light Fuel Oil (Diesel Oil and Kerosene)

The oncogenic potential of petroleum fuels is directly related to refinery processing methods used to obtain the petroleum product and the crude oil composition from which the fuel was derived. An evaluation of the composition of petroleum fuels has revealed that a positive correlation exists between polycyclic aromatic hydrocarbon (PAH) content and carcinogenicity in human epidemiology studies or experimental laboratory studies (Bingham et al., 1979).

Diesel fuel is usually a straight-run distillation product composed of a complex variable mixture of hydrocarbons with a boiling point range of 175 to 370 °C (DOE, 1983). Although the aromatic content ranges to 35 percent, few of them are polycyclic compounds. Diesel fuel has not been shown to be carcinogenic. In a 2-year oncogenic skin painting study, which was terminated after 62 weeks because of the presence of extensive skin lesions, Swiss Epley mice were exposed to 0.05 ml (41 mg) of diesel fuel products. Skin carcinomas were found in 2 of 50 animals, which was not statistically significant by chi-square analysis (American Petroleum Institute, 1983b).

Kerosene is a straight-run distillation product with a boiling point range of 175 to 325 °C (HSDB, 1987a) and an aromatic content of 18 percent (Conaway et al., 1982). Higher boiling point (greater than 370 °C) petroleum products that are subjected to additional refinement processes, such as cracking or hydrogenation, and that contain polycyclic aromatics may be carcinogenic to experimental animals (Bingham et al., 1979).

Specific substances that are known or suspected of being carcinogenic, which are contained in diesel oil and kerosene in small amounts, include benzo(a)pyrene and benzene (Bingham et al., 1979). Benzo(a)pyrene (BaP), a potent carcinogen, is a PAH that also occurs at low levels in foods and in products of combustion, including cigarette smoke (Bingham et al., 1979). Bioassays indicate that the concentration of this single carcinogen can often serve as a guide in predicting carcinogenic potency, although other substances are also known to be involved (Bingham et al., 1979). There is sufficient evidence to conclude that BaP is carcinogenic in experimental animals: BaP has incited tumors in all of the nine species for which data have been reported, despite the use of different methods of administration (U.S. Department of Health and Human Services (DHHS), 1985). These studies reported both local and systemic carcinogenic effects.

For benzene, another aromatic hydrocarbon known to be present in petroleum fuels, there is sufficient evidence to indicate that it is carcinogenic in experimental animals and in humans (U.S. DHHS, 1985). Benzene has been shown to cause leukemia in chronically exposed workers (U.S. DHHS, 1985).

Because of the carcinogenicity of the aromatic hydrocarbons found in diesel fuel and kerosene, these light fuel oils are considered carcinogenic for this risk assessment.

Limonene

No chronic studies have been reported for limonene. However, studies have indicated regression and inhibition of tumor growth following dietary administration of d-limonene (Elegbede et al., 1986a; Elegbede et al., 1986b; Van Duuren and Goldschmidt, 1976). There are insufficient data to determine the cancer risk for limonene in this risk assessment.

Picloram

There has been disagreement among experts on the interpretation of studies about the potential of picloram to cause cancer. A rat oncogenicity study, in which test animals were exposed to an average of 743 mg/kg/day, was reported to be negative for oncogenic effects in males. However, benign liver tumors (nodules) were observed in females (EPA, 1984f). A recently reported 2-year rat chronic toxicity-oncogenicity study observed no treatment-related increases in tumor incidence at any dose level (20, 60, or 200 mg/kg/day) (Dow, 1987a). A mouse oncogenicity study showed no tumor formation at dietary exposure levels ranging from 5,000 to 15,000 ppm (750 mg/kg to 2,250 mg/kg) (EPA, 1984f). Because of the female rat results, a cancer risk analysis will be conducted on picloram in this risk assessment as if picloram is carcinogenic.

Sulfometuron Methyl

No oncogenic effects were reported from a 2-year rat feeding or the 1-year chronic dog feeding studies (DuPont, 1986). Based on these data, sulfometuron methyl is not considered carcinogenic for this risk assessment.

Tebuthiuron

Available evidence does not indicate that tebuthiuron is carcinogenic. In 2-year mouse and rat feeding studies, no oncogenic effects of tebuthiuron were observed up to 240 and 400 mg/kg/day (HDT, respectively) (EPA, 1986e). For the purpose of this risk assessment, tebuthiuron is considered nononcogenic.

Triclopyr

Available data do not indicate that triclopyr is carcinogenic. For both rat and mouse 2-year feeding studies, no oncogenic effects were apparent in test animals exposed to triclopyr (30 and 36, respectively) (EPA, 1986f; 40 CFR Part 180 50(84):184-85, May 1, 1985). A recent 2-year chronic toxicity-oncogenicity study in rats has been submitted in response to EPA's request for a repeat rat oncogenicity study (Dow, 1987a). A statistically significant increase in mammary tumors was observed when the number of adenomas (1) and adenocarcinomas (4) were combined for high dose females (36 mg/kg/day) (Dow, 1987a). However, the researchers reported that the incidence was within a range of historical controls and the statistical result was partially because of the low incidence (0) in control rats. Based on these results, triclopyr is not considered carcinogenic for this risk assessment.

CANCER POTENCY

This subsection presents the results of the cancer potency analysis for each of the herbicides assumed to be carcinogenic in this risk assessment. The cancer potency value is used later in the risk analysis to determine the human cancer risk under specified assumptions about lifetime human exposure.

The cancer potency of a chemical is defined as the increase in likelihood of getting cancer from a unit increase in the dose of the chemical. An example of this relationship is illustrated by the graph in figure 3-2. The slope of the line specifies what the increase in cancer probability is for each unit increase in dose in mg/kg/day. The cancer potency value reflects the probability of getting cancer sometime in a person's lifetime for each mg/kg/day.

The cancer potency is derived from tumor data generated in laboratory animal studies. Note in figure 3-2 that the dose levels used in the laboratory cancer studies are high, but those that humans are likely to experience from exposure to the environment are low. Note also that the line relating dose to cancer probability approximates a straight line in the low dose region.

Several assumptions have been made in estimating cancer potencies. First, it is assumed that any dose, no matter how small, has some probability of causing cancer. This is an assumption based on the nonthreshold hypothesis, discussed previously, which postulates that even a single, extremely small dose may be enough to trigger cancer. Second, one of the principal areas of scientific controversy in cancer risk assessment is extrapolating from the high doses used in animal studies to the far lower

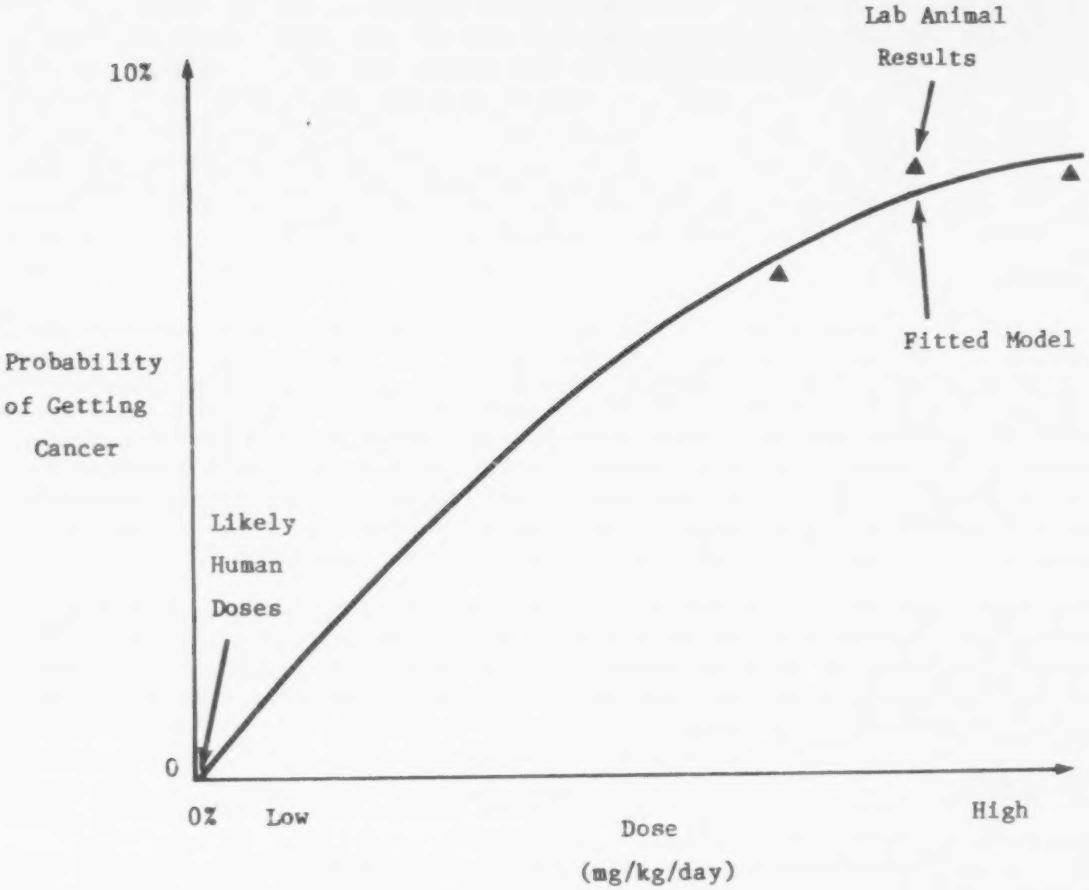

Figure 3-2--Cancer potency curve

doses humans may get. Models other than the linearized multistage model, which assumes a straight line at low doses, as illustrated in figure 3-2, have been used for the extrapolation of cancer data to assess human risk. However, this model is believed to be reasonably conservative (not underestimating risk), and it is the model currently used by EPA. Cancer potency values reported by EPA were used for benzene and BaP. Third, the cancer potency used in the calculation of human risk in this analysis is not the maximum likelihood potency value, but the upper limit value of the 95-percent statistical confidence interval.

2,4-D

2,4-D cancer potency was calculated based on the rate of tumor formation in the female Osborne-Mendel rats studied by Hansen et al. (1971). This is the species and sex that have exhibited the greatest increase in tumor formation after 2,4-D administration. All tumors were considered, although many of them were benign. The conservative one-hit model was used to represent the relationship between dose and rate of tumor formation. The 95-percent upper confidence limit of the cancer potency, calculated by Crump (1983) using the GLOBAL 82 computer program, was 0.00503 per (mg/kg/day). EPA (1986m) has stated that their preliminary review of an additional long-term oncogenicity study submitted to EPA indicates that the cancer potency level would be of about the same magnitude as the cancer potency calculated by Crump.

2,4-DP

A cancer study involving rats fed up to 200 mg/kg (EPA, 1982b) was used to derive 2,4-DP cancer potency. In this study, the highest dose group showed signs of general toxicity because they were fed more than the maximum tolerated dose of 2,4-DP. Many of the females at all dose levels had tumors, but they did not show a dose-related response. The high dose group actually had fewer malignant tumors than the intermediate dose group. Males showed a significant increase in the incidence of malignant tumors, with a corresponding decrease in the incidence of benign tumors. The tumors were primarily in the thyroid and pituitary glands.

The 95-percent upper confidence limit for the cancer potency of 2,4-DP was estimated from the male rat data as 0.0124 per (mg/kg/day). Only malignant tumors were considered in this case, and the high dose group showing signs of general toxicity was not considered in order to give the highest cancer potency indicated by the data.

Glyphosate

Although glyphosate has not been shown to be a carcinogen, an upper limit for its cancer potency was estimated based on the rate of benign kidney tumor formation in male mice in the feeding study reported in EPA (1985d). Controversy exists over whether glyphosate should be classed into EPA category C (possible carcinogen) or category D (data insufficient to classify).

Because of the lack of conclusive data, a conservative approach was adopted in this risk assessment, and glyphosate's carcinogenic potential was evaluated. The upper 95-percent limit of the cancer potency of glyphosate calculated from the kidney tumor data was 0.000026 per (mg/kg/day).

Light Fuel Oil (Diesel Oil and Kerosene)

The carcinogenic potencies of diesel oil and kerosene have been estimated for this risk assessment based on the potencies of both benzene and BaP. EPA (1986n) has estimated the carcinogenic potency of BaP as 11.5 per (mg/kg/day).

The carcinogenic potency of benzene, however, is much less than that of BaP. EPA has estimated the carcinogenic potency of benzene as 0.0445 per (mg/kg/day) (EPA, 1986o).

Samples of diesel oil and fuel oil have been found to have a BaP content of only 0.026 ppm, but No. 2 heating oil (which may be subjected to cracking, rather than being a straight-run distillation product) can contain 600 ppb (Bingham et al., 1979). The midpoint of this concentration range (313 ppb) has been used to calculate the carcinogenic potency of diesel oil, although most diesel fuels can be expected to have a lower BaP content. The content of benzene in diesel fuel was assumed to be 28.5 ppm, based on analysis of water extracts of No. 2 fuel oil by Anderson (1975), with corrections for solubility relationships. The resulting estimate of carcinogenic potency of diesel oil is 0.0000049 per (mg/kg/day). Seventy-four percent of this potency is a result of the BaP component.

Picloram

The Gulf Research Institute conducted a carcinogenic bioassay of picloram in rats and mice for the National Cancer Institute (1978). There was evidence that picloram affected the livers of male and female rats, and the study concluded that the findings were "suggestive of ability of the compound to induce benign tumors in livers of female Osborne-Mendel rats." Currently, picloram is classified by EPA in category D, meaning that the data are insufficient to classify it as to carcinogenicity.

Because of the weakly positive response in the 1978 NCI study, a conservative approach was adopted in this risk assessment, and the carcinogenic potential of picloram is evaluated. Using this study, the upper 95-percent confidence limit on the cancer potency of picloram has been calculated to be 0.00057 per (mg/kg/day) using the GLOBAL 82 computer program (Crump, 1983).

INERT INGREDIENTS

Inert ingredients are chemicals used with the active ingredient in preparing herbicide formulation. They are used to provide a carrier for the active ingredient that facilitates the effective application of the herbicide. Inerts are not intended to supplement the herbicide's toxic properties. Table 3-7 lists the percentage of inert ingredients in herbicides being evaluated for use in Region 8.

This risk assessment characterizes human health risks by comparing estimated herbicide doses with toxicity levels found in laboratory animal studies. The estimated doses and laboratory hazard levels are based on the active ingredients of the proposed herbicides, not on the formulated products. This is reasonable because the active ingredients possess the intended pesticidal properties. However, consideration of the possible toxic properties of the remaining portion of the formulations, the inert ingredients, is also warranted as is the possibility of synergism from the combination of active and inert ingredients in the formulations.

EPA (1987d) noted that concerns regarding the acute toxicity of inert ingredients are usually addressed through tests of the herbicides as formulated products. While the herbicides as formulated products have

Table 3-7

Percentage of inert ingredients present in herbicide formulations used in Region 8

Chemical	Formulation	Percent Inerts
2,4-D	Esteron 99®	37.2
	Weed Rhap A-4D®	53.26
	Weedar 64®	53.2
2,4-DP	Weedone®	36.3
	Weedone CB®	76.4
Dicamba	Banvel®	43.1 (100% water)
	Banvel 720®	59.5 (100% water)
	Banvel CST®	72.4
Fosamine	Krenite®	58.5 (89% water)
	Krenite S®	58.5 (70% water)
Glyphosate	Roundup ®	59 (85% water)
	Rodeo®	46.5 (100% water)
	Accord®	59 (100% water)
Hexazinone	Velpar L®	75
	Pronone 10G®	90
	Pronone 5G®	95
Imazapyr	Arsenal®	72.4
Picloram	Tordon 101®	50.2
	Tordon 101R®	73.7
Sulfometuron methyl	Oust®	25
Tebuthiuron	Spike 40®	60
	Spike DF®	15
	Spike 5G®	95
Triclopyr	Garlon 3A®	55.6
	Garlon 4®	38.4

Source: Pesticide labels.

undergone acute toxicity testing, they generally have not undergone extensive chronic toxicity testing, or cancer, reproductive, developmental, or mutagenicity testing. The gap in the testing of the herbicides as formulated products, according to one view, gives rise to the inference that the environmental consequences, including hazards to human health, from using them are largely unknown. The hypothesis holds that regardless of what is known about a herbicide formulation's two components (the active ingredients and inerts), the possibility exists that the formulated product may pose a greater or lesser risk (due to synergism or antagonism) than separate consideration of each component may suggest. (Refer to the Synergistic Effects section of this risk assessment for a more detailed discussion of synergism.) Given the small amount of information that is available on each herbicide's formulation, this possibility cannot be discounted entirely, neither can it be presumed to be true. The possibility that herbicidal formulations may pose greater risk than their components is largely an untested hypothesis, and where acute toxicity data are available for herbicidal formulations, this hypothesis has been disproven.

An alternate viewpoint, the one adopted in this risk assessment, is that the data gaps about the herbicides as formulated products are not a primary concern because the risks posed by the herbicides' active ingredients are overstated. Any risk posed by the herbicides as formulated products is considered to be characterized by the analysis of the active ingredients. The herbicides' active ingredients have undergone cancer, reproductive, developmental, and mutagenicity tests of varying degrees. The herbicides' inerts have undergone categorization according to their suspected toxicity and predicted risks. With only one exception, kerosene, which is being addressed in this risk analysis, no specific concern exists with the herbicides' inerts. Thus, because the herbicides' active ingredients here, not their inerts, are the source of toxicity, it logically follows that any analysis drawing attention to the former as opposed to the latter is properly focused.

Toxicity of the Inert Ingredients

With respect to the toxicity of the inert ingredients alone, EPA's Office of Pesticide Programs (EPA, 1986p) has identified about 1,200 inert ingredients that are now used in approved pesticides and has reviewed the available evidence concerning their toxicity. The data included laboratory toxicity tests, epidemiological data, and structure/activity relationships. A particular concern in reviewing the inerts was their potential for causing chronic human health effects. On completion of its review, EPA categorized the 1,200 inerts into four lists.

> List 1 contains about 55 inerts that have been shown to be carcinogens, developmental toxicants, neurotoxins, or potential ecological hazards and that merit the highest priority for regulatory action.
>
> List 2 contains approximately 50 inerts that have been given high priority for testing because toxicity data are suggestive, but not conclusive, of possible chronic health effects or because they have structures similar to chemicals on List 1.

List 3 contains about 800 inerts that are of lower priority because no evidence from toxicity data or from a review of their chemical structure would now support a concern for toxicity or risk.

List 4 of about 300 inerts contains those inerts generally recognized as safe.

Because EPA normally classifies inert ingredients as "Confidential Business Information," information on them does not have to be released by EPA to the public under the Freedom of Information Act. (See also 40 CFR 1506(a).) Nonetheless, the Forest Service requested that EPA review the herbicides proposed for use and disclose whether any of them contain inert ingredients of or suggesting toxicological concern. EPA has completed this review for some of the chemical formulations and is currently reviewing the remaining formulations. EPA will inform the Forest Service when the review is complete. The Forest Service has also requested information on inerts from the chemical companies that manufacture the herbicides. The chemical companies have voluntarily submitted this information to the Forest Service.

So far, EPA and the chemical companies have identified only one inert ingredient on either List 1 or List 2 (see table 3-8). This ingredient is kerosene, which is considered a "petroleum hydrocarbon"; it is on List 2, and therefore has high priority for testing. Kerosene is used as a solvent in a number of formulations that contain 2,4-D, triclopyr, and picloram (Dow, 1987b). The human health risk from exposure to kerosene in such products is estimated in the exposure and risk analyses in sections 4 and 5 of this risk assessment. The Forest Service will continue to monitor the status of inert ingredients in the formulations they use and will do further assessments and revisions if they are recategorized.

Toxicity of the Formulations

With respect to the possibility of synergism in the formulated combination of active and inert ingredients, EPA generally requires only acute toxicity data on formulated products. These data also allow EPA to address concerns about the acute toxicity of the pesticide formulations' inert ingredients. A comparison of their acute LD_{50}'s provides an indication of the toxicity of the formulated product (including inerts) versus the active ingredient alone. As shown in table 3-9, the formulations proposed for use by the Forest Service are less acutely toxic than their active ingredient.

Table 3-8

Toxicity of identified inert ingredients of Region 8 chemical formulations

Inert Ingredients	Toxicity
Aliphatic alcohol	List 4 inert; very slightly toxic; eye irritant
Aryl sulfonate (detergent)	List 3 inert; slightly to very slightly toxic; irritant
Aryl sulfonate polymer	List 3 inert; very slightly toxic; slight irritant
Blend of amine alkylbenzene sulfonates (containing petroleum distillates and n-butanol)	List 3 inerts; no specific toxicity data available
Chelating agent	List 3 inert; slight toxicity; slight irritant
Clay carrier	List 4 inert; generally recognized as safe
Ethylene glycol	List 3 inert; moderately toxic; irritant
Inorganic salt (buffer)	List 4 inert; slightly toxic; irritant
Kerosene	List 2 inert; LD_{50} >28,000 mg/kg
Nonionic surfactant	LD_{50} = 8.2 ml/kg; list category not given
Organic ether polymer	List 3 inert; very slightly toxic
Polyethoxyethylene ester	List 3 inert; no toxicity data available, but only very slight toxicity expected due to its chemical nature
Polyethoxyethylene ether	List 3 inert; slightly toxic; slight irritant
Polyethoxylated tallow amine	List 3 inert; oral LD_{50} = 1,200 mg/kg/day dermal. LD_{50} >1,260 mg/kg; negative for irritation and sensitization in humans for 30-percent solution
Polyglycol	List 3 inert; slightly toxic; slight irritant
Water	List 4 inert; generally recognized as safe

Source: Dow Chemical Company, Monsanto Company, DuPont Chemical Company, American Cyanamid Company, Pro-Serve, Inc.

Table 3-9

Technical grade and formulation
acute oral LD_{50} values for rats

Herbicide	Technical Grade Acute Oral LD_{50} Values for Rats	Formulation Acute Oral LD_{50} Values for Rats
2,4-D	375 mg/kg (2,4-D acid) (EPA, 1986L)	Esteron 99 (Butoxyethyl Ester)-- 25,000 mg/kg, males tested: 21,000 mg/kg, females tested (Vertac, 1982) Weedar 64 (Dimethylamine salt)-- 1615 ± 170 mg/kg males tested (Vertac, 1977)
2,4-DP	532 mg/kg (EPA, 1984a)	Weedone-- 2,200 ± 350 mg/kg (EPA, 1984a)
Fosamine	NA[a]	Krenite-- 24,400 mg/kg (USDA, 1984) Krenite-- >5,000 mg/kg (USDA, 1984)
Glyphosate	4,320 mg/kg (EPA, 1986c)	Roundup-- 4,900 to 5,400 mg/kg (USDA, 1984) Rodeo-- >5,000 mg/kg (Monsanto, 1983)
Hexazinone	1,690 mg/kg (EPA, 1986d)	Velpar L-- 6,887 mg/kg (DuPont, 1985) Pronone 5G-- >5,000 mg/kg (DuPont, 1984b) Pronone 10G-- >5,000 mg/kg (DuPont, 1984b)
Imazapyr	>5,000 mg/kg (EPA, 1985b)	NA
Limonene	NA	Cide-Kick-- >5,000 mg/kg (HSDB, 1987b)
Picloram	8,200 mg/kg (EPA, 1984c)	Tordon 22K[b]-- 8,440 mg/kg (Mullison, 1985)
Sulfometuron methyl	>5,000 mg/kg (DuPont, 1983b)	Oust-- >5,000 mg/kg (DuPont, 1983b)

Table 3-9 (continued)

Technical grade and formulation acute oral LD_{50} values for rats

Herbicide	Technical Grade Acute Oral LD_{50} Values for Rats	Formulation Acute Oral LD_{50} Values for Rats
Triclopyr	630 mg/kg, females tested; 729 mg/kg, males tested (USDA, 1984)	Garlon 3A-- 2,140 mg/kg, females tested; 2,830 mg/kg, males tested (Dow, 1986a) Garlon 4-- 2,140 mg/kg, females tested; 2,460 mg/kg, males tested (Dow, 1986a)

[a] Not available.

[b] Tordon 22K will not be used in Region 8, but was included for comparison.

Section 4

HUMAN EXPOSURE ANALYSIS

This chapter presents the background, methods, and some results of the herbicide exposure analysis. The first section contains basic background information used in defining the exposure analysis methods. Some terminology relating to herbicide use and potential human exposure is discussed. Subsequent sections contain descriptions of herbicide usage in vegetation management operations and the potential routes of human exposure in those operations. The methods used to estimate herbicide doses to workers and members of the public also are discussed. Lifetime dose estimates are also presented as preliminary steps in the estimation of lifetime cancer risks. The exposures have been calculated for typical and maximum exposure situations. Representative doses calculated in the exposure analysis will be presented in section 5 in conjunction with the margins of safety.

BACKGROUND

This section defines some of the terms used in the discussion of exposure analysis methods and results. Potential routes of human exposure are also explained.

Herbicide Characteristics

Most herbicides used in the Southern Region are formulated and sold by the manufacturer as solutions or granules. Herbicides sold in liquid form are sold as concentrates with a specified number of pounds of active ingredient, usually between 1 and 4, per gallon of concentrate, and with inert ingredients forming the remaining portion. Herbicide concentrates are normally stored and transported in 5-gal (or smaller) containers. Granular material is transported and stored in 50- to 100-lb bags.

Before liquid herbicides are applied, they are mixed with a carrier, usually water, according to the manufacturer's label instructions for the particular treatment purpose and the desired application rate, which may be at or below the labeled use rate. For ground broadcast operations, the concentrate is typically mixed with up to 25 gal of water for every acre to be treated. Aerial applications require less water, typically 10 to 15 gal/ac. Limonene and/or light fuel oil (usually diesel fuel) may be added to the mixture in either aerial or ground broadcast applications. Soil spot applications are usually done with a 1:2 or 1:3 water dilution. Cut surface applications are normally done with formulations that are undiluted or diluted 1:2 or 1:3. Basal bark/stem applications may be done with 1 to 2 gal/ac of diesel fuel used as a carrier; or limonene may also be used as an adjuvant.

Herbicide spray application equipment is designed to treat the target plants or soil with a minimum of off-target movement of airborne spray droplets, called drift. Spray nozzles used in the Region are designed to produce large droplets because smaller droplets tend to remain airborne and may drift with air currents away from the target vegetation. Despite the effectiveness of the spray application equipment used, some small fraction of the droplets may break up into smaller droplets that the wind could blow offsite.

Hand application equipment used for soil spot, streamline, basal bark/stem, and hack and squirt techniques do not produce spray but rather a directed stream of formulation. Thus, these techniques do not produce herbicide drift. The potential for drift of granular formulations also is negligible, although some dust may be encountered during handling and application.

Exposure and Dose

Two primary conditions are necessary for a human to receive a dose of herbicide that may result in a toxic effect. First, the herbicide must be present in the person's immediate environment so that it is available for intake. It must be in the air the person breathes, or on the person's skin, or in the person's food or water. The amount of herbicide present in the person's immediate environment is the exposure level.

Second, the herbicide must move into the person's body. If it is in the air, it must be inhaled into the air passages and lungs. If it is on the clothing or skin, it must penetrate the skin. If it is in food or water, it must be ingested. The amount that moves into the body by any of these routes constitutes the dose.

Thus, although two people may be subjected to the same level of exposure, one may get a much lower dose than the other by wearing protective clothing, using a respirator, or washing immediately after spraying. Exposure, then, is the amount of pesticide available to be taken in; dose is the amount that actually enters the body.

EXPOSURE ANALYSIS METHODS

This section describes how herbicide doses were calculated for members of the public and workers. The data, assumptions, and methods of calculation are presented, and some of the factors affecting the magnitude of the doses are discussed. A set of example exposure situations is chosen as a basis for the risk calculations presented in section 5.

Exposure Scenarios

Region 8 vegetation management personnel were consulted to obtain realistic estimates of several important factors relating to herbicide application practices. The acreage of National Forest land that is treated with herbicides for various purposes are shown in table 4-1. Most of the herbicides have been used for a variety of purposes. Tebuthiuron was used only for right-of-way applications. The use of tebuthiuron on rights-of-way is expected to continue in the future, but no other uses of tebuthiuron are anticipated.

Table 4-1

Acreage of treatment operations by herbicide for Region 8 lands in 1986[a]

Chemical	Conifer Release	General Weeds	Hardwood Release	Noxious Weeds and Poisonous Plants	Range Improvement	Right-of-Way	Site Preparation	Thinning	Wildlife Habitat Improvement
2,4-D amine	1300	25	900	300	400	350	6000	2100	1050
2,4-D ester	0	+[b]	0	0	500	50	250	0	125
2,4-DP	4300	0	0	0	0	50	10750	400	50
Dicamba	75	+	0	75	50	50	1100	25	0
Fosamine	0	+	0	0	0	125	0	0	0
Glyphosate	3750	75	1175	325	50	100	5225	0	50
Hexazinone	25675	0	500	0	100	375	19600	800	525
Imazapyr	0	0	0	0	0	0	0	0	0
Picloram	725	+	725	200	50	0	4875	600	600
Sulfometuron methyl	1225	1125	0	0	0	50	325	0	0
Tebuthiuron	0	0	0	0	0	150	0	0	0
Triclopyr amine	3550	0	350	0	50	25	5800	275	100
Triclopyr ester	7750	0	350	0	0	175	2700	225	100

[a] Numbers are rounded to the nearest 25. Totals for herbicide usage by treatment objective will not necessarily agree with those presented in table 1-1 because many acres would be double counted. Double counting occurs when herbicides are used in combination either as a tank mix or formulated product. For example, Tordon is a combination of 2,4-D and picloram, so Tordon treated acreage is listed under both herbicides.

[b] + = Incidental usage on less than 12.5 acres.

Source: USFS Region 8, 1987.

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The potential for exposure to the herbicides depends primarily on the manner and place of application; the purpose of the application has only an indirect influence. Consequently, most of the data needed for the exposure analysis were collected according to the application method. These data and the subsequent exposure calculations are intended to represent two basic cases: typical and maximum. The maximum case shows the highest exposures anticipated under realistic application conditions.

The typical and maximum number of acres expected to be treated annually with each herbicide are shown in table 4-2 for each application method. The annual number of acres treated shows the relative importance of the application methods, but it is desirable to calculate exposures on a per-day basis. Doses calculated per day are consistent with standard toxicity reference levels, such as the NOEL, which are usually expressed in per-day units. The typical and maximum number of acres expected to be treated at a time are shown in table 4-3. The typical and maximum number of hours per day expected to be worked on each type of application are shown in table 4-4. The maximum number of hours is in some cases greater than 8, ranging as high as 11.

It should be noted that the time period specified refers to total project time, not time of mixing, loading, or application. The use of total project time is consistent with times commonly given in worker exposure studies, for example, Lavy et al. (1982).

Consideration of daily exposure is adequate for evaluation of the risk of threshold effects, but cumulative exposures must be considered in order to evaluate the risk of cancer. The typical and maximum number of potential exposure days per year have been estimated for a single worker, and they are presented in table 4-5.

The rate of application, in terms of pounds of active ingredient per acre, is expected to have a direct relationship with most of the potential types of exposure. The typical and maximum anticipated application rates (lb/ac a.i.) are shown in table 4-6 for each herbicide. Smaller amounts are used if they are found to be effective, for purposes of economy and safety. The Forest Service usually uses less than the maximum allowable rate specified by the EPA registration. (Application rates are presented in units of lb/acre for comparison with registration and label information, but all exposure calculations will be presented in metric units.)

The potential routes of human exposure considered in this risk assessment are outlined in table 4-7 and are described below. These routes of exposure are considered in estimating doses to the public and workers that might occur during routine operations or in the event of an accident. The greatest doses to humans during routine herbicide applications are to workers who may be exposed while (1) mixing and loading herbicide into application equipment, (2) applying herbicide to vegetation using ground-based equipment, or (3) supervising or monitoring aerial or ground-based herbicide applications. Workers may be dermally exposed to an herbicide if the herbicide concentrate, mixture, or drifting spray droplets contact the skin or if the herbicide is brushed off sprayed vegetation. Inhalation exposure may result from breathing without protective devices in

Table 4-2

Typical and maximum anticipated number of acres treated per year in Region 8[a]

Application Method	2,4-D amine	2,4-D ester	2,4-DP	Dicamba	Fosamine	Glyphosate	Hexazinone	Imazapyr[b]
Aerial[b]								
Foliar	600 [600]	90 [100]	85 [100]		400 [1000]	1300 [2000]	580 [1200]	200 [1500]
Granular/pellets							1500 [6000]	
Mechanical								
Foliar	700 [700]	100 [300]	250 [300]	500 [100]	100 [250]	3000 [7000]	4200 [1800]	400 [2000]
Granular/pellets							6400 [7800]	
Manual Ground								
Application method Granular/pellets							600 [1100]	
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	2800 [4700]	600 [700]	15500 [11700]	800 [200]		1300 [6200]	3500 [5000].	200 [1000]
Basal bark/stem		200 [200]	900 [1100]					
Basal soil/soil spot							32500 [40600]	
Cut surface (frill, injection, cut stump, hack and squirt, etc.)	6600 [9500]			50 [100]		4100 [3900]		800 [2000]

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[a] Totals for herbicide usage by type of treatment objective will not necessarily agree with those presented in table 2-1 since many acres would be double counted. Double counting occurs when herbicides are used in combination either as a tank mix or formulated product with either another herbicide or with an additive (example Tordon is a combination of 2,4-D and Picloram and all of the Tordon treated acreage is presented in the treatment acreage totals for both herbicides). Limonene and light fuel oils have been excluded from acreage sums since they are always used as additives in herbicide applications and would, therefore, always be double counted.

[b] Values are estimated; chemical or application method not currently used.

[ ]--Maximum anticipated.

Table 4-2 (continued)

Typical and maximum anticipated number of acres treated per year in Region 8[a]

Application Method	Light Fuel Oil	Limonene	Picloram	Sulfometuron Methyl	Tebuthiuron[b]	Triclopyr Amine	Triclopyr Ester
Aerial[b]							
Foliar	100 [1500]	700 [3000]	200 [250]	70 [200]	200 [1000]	130 [500]	100 [1500]
Granular/pellets					300 [1500]		
Mechanical							
Foliar	2000 [5000]	3400 [9000]	350 [500]	300 [800]	50 [500]	50 [100]	2400 [5600]
Granular/pellets					100 [1000]		
Manual Ground							
Application method							
Granular/pellets							
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	3600 [5500]	8200 [19200]	500 [100]	2100 [2300]	400 [1000]	250 [2600]	2700 [15000]
Basal bark/stem	4500 [7500]	1600 [5200]					7800 [12500]
Basal soil/soil spot					400 [1000]		
Cut surface (frill, injection, cut stump, hack and squirt, etc.)			7200 [2000]			9600 [10400]	

[a] Totals for herbicide usage by type of treatment objective will not necessarily agree with those presented in table 2-1 since many acres would be double counted. Double counting occurs when herbicides are used in combination either as a tank mix or formulated product with either another herbicide or with an additive (example Tordon is a combination of 2,4-D and Picloram and all of the Tordon treated acreage is presented in the treatment acreage totals for both herbicides). Limonene and light fuel oils have been excluded from acreage sums since they are always used as additives in herbicide applications and would, therefore, always be double counted.

[b] Values are estimated; chemical or application method not currently used.

[ ]—Maximum anticipated. The numbers presented in this column are field estimates based on current herbicide use rates. They were made prior to the scoping processes for two of the three EIS's to which this risk assessment will tier. They may not reflect alternatives proposed subsequently but are used as current best-estimates for computation purposes only.

Table 4-3

Typical and maximum anticipated number of acres treated in a single project in Region 8

Application Method	2,4-D amine	2,4-D ester	2,4-DP	Dicamba	Fosamine	Glyphosate	Hexazinone	Imazapyr[a]
Aerial[a]								
Foliar	20 [160]	15 [150]	12 [160]		18 [150]	30 [400]	30 [180]	60 [400]
Granular/pellets							60 [400]	
Mechanical								
Foliar	40 [100]	30 [80]	40 [80]	40 [100]	10 [100]	30 [400]	55 [500]	55 [500]
Granular/pellets							55 [550]	
Manual Ground								
Application method Granular/pellets							39 [300]	
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	25 [100]	16 [80]	60 [100]	25 [100]		20 [80]	60 [100]	60 [100]
Basal bark/stem		5 [10]	15 [65]					
Basal soil/soil spot							42 [2000]	
Cut surface (frill, injection cut stump, hack and squirt, etc.)	28 [100]			5 [10]		30 [100]		10 [80]

[ ]--Maximum anticipated

[a]Values are estimated; chemical or application method not currently used.

Table 4-3 (continued)

Typical and maximum anticipated number of acres treated in a single project in Region 8

Application Method	Light Fuel Oil	Limonene	Picloram	Sulfometuron Methyl	Tebuthiuron[a]	Triclopyr Amine	Triclopyr Ester
Aerial[a]							
Foliar	60 [100]	60 [200]	20 [160]	20 [150]	120 [400]	18 [160]	50 [600]
Granular/pellets					150 [500]		
Mechanical							
Foliar	60 [100]	50 [625]	40 [100]	33 [80]	40 [100]	100 [500]	50 [625]
Granular/pellets					50 [200]		
Manual Ground							
Application method Granular/pellets							
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	60 [100]	60 [100]	30 [75]	27 [80]	40 [100]	20 [60]	20 [100]
Basal bark/stem	27 [100]	27 [100]					25 [100]
Basal soil/soil spot					40 [100]		
Cut surface (frill, injection, cut stump, hack and squirt, etc.)			50 [120]			20 [80]	

[ ]--Maximum anticipated

[a]Values are estimated; chemical or application method not currently used.

Table 4-4

Typical and maximum anticipated worker exposure in hours per day in Region 8

Application Method	2,4-D amine	2,4-D ester	2,4-DP	Dicamba	Fosamine	Glyphosate	Hexazinone	Imazapyr[a]
Aerial[a]								
Foliar	4 [8]	4 [8]	4 [8]		4 [8]	4 [8]	4 [8]	8 [8]
Granular/pellets							8 [8]	
Mechanical								
Foliar	6 [11]	5 [7]	6 [8]	6 [10]	5 [7]	6 [8]	6 [8]	6 [8]
Granular/pellets							6 [9]	
Manual Ground								
Granular/pellets							6 [8]	
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	6 [8]	5 [6]	6 [10]	6 [10]		6 [8]	6 [10]	6 [10]
Basal bark/stem		6 [6]	6 [10]					
Basal soil/soil spot							6 [8]	
Cut surface (frill, injection, cut stump, hack and squirt, etc.)	6 [8]			6 [10]		6 [8]		6 [7]

[ ]--Maximum anticipated

[a] Values are estimated; chemical or application method not currently used.

Table 4-4 (continued)

Typical and maximum anticipated worker exposure in hours per day in Region 8

Application Method	Light Fuel Oil	Limonene	Picloram	Sulfometuron Methyl	Tebuthiuron[a]	Triclopyr Amine	Triclopyr Ester
Aerial[a]							
Foliar	5 [7]	3 [8]	4 [8]	4 [8]	3 [8]	4 [8]	5 [8]
Granular/pellets					6 [8]		
Mechanical							
Foliar	6 [11]	6 [10]	8 [11]	6 [8]	6 [11]	7 [8]	7 [8]
Granular/pellets					6 [11]		
Manual Ground							
Granular/pellets							
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	6 [10]	5 [10]	6 [8]	6 [7]	5 [8]	4 [6]	4 [6]
Basal bark/stem	6 [8]	6 [8]					6 [8]
Basal soil/soil spot					5 [8]		
Cut surface (frill, injection, cut stump, hack and squirt, etc.)			7 [10]			5 [9]	

[ ]--Maximum anticipated

[a] Values are estimated; chemical or application method not currently used.

Table 4-5

Typical and maximum anticipated worker exposure in days per year in Region 8

Application Method	2,4-D amine	2,4-D ester	2,4-DP	Dicamba	Fosamine	Glyphosate	Hexazinone	Imazapyr[a]
Aerial[a]								
Foliar	9 [12]	3 [5]	3 [5]		6 [15]	2 [20]	8 [18]	1 [9]
Granular/pellets							4 [20]	
Mechanical								
Foliar	15 [15]	5 [15]	15 [25]	10 [30]	3 [5]	11 [20]	22 [26]	8 [52]
Granular/pellets							23 [34]	
Manual Ground								
Application method								
Granular/pellets							14 [17]	
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	25 [49]	11 [25]	75 [90]	10 [40]		13 [80]	14 [40]	14 [15]
Basal bark/stem		5 [15]	18 [35]					
Basal soil/soil spot							33 [46]	
Cut surface (frill, injection, cut stump, hack and squirt, etc.)	38 [57]			14 [40]		32 [60]		56 [105]

[ ]--Maximum anticipated

[a] Values are estimated; chemical or application method not currently used.

Table 4-5 (continued)

Typical and maximum anticipated worker exposure in days per year in Region 8

Application Method	Light Fuel Oil	Limonene	Picloram	Sulfometuron Methyl	Tebuthiuron[a]	Triclopyr Amine	Triclopyr Ester
Aerial[a]							
Foliar	2 [15]	10 [30]	3 [5]	1 [3]	2 [10]	2 [8]	2 [10]
Granular/pellets					4 [15]		
Mechanical							
Foliar	30 [70]	100 [160]	10 [15]	10 [30]	3 [5]	5 [10]	8 [15]
Granular/pellets					2 [10]		
Manual Ground							
Granular/pellets							
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	50 [80]	200 [270]	15 [25]	16 [19]	5 [10]	8 [15]	20 [40]
Basal bark/stem	33 [42]	33 [46]					33 [42]
Basal soil/soil spot					5 [10]		
Cut surface (frill, injection, cut stump, hack and squirt, etc.)			36 [64]			45 [50]	

[ ]--Maximum anticipated

[a] Values are estimated; chemical or application method not currently used.

Table 4-6

Typical and maximum anticipated application rates in pounds per acre a.i. in Region 8

Application Method	2,4-D amine	2,4-D ester	2,4-DP	Dicamba	Fosamine	Glyphosate	Hexazinone	Imazapyr[a]
Aerial[a]								
Foliar	2.0 [4.0]	2.5 [6.0]	3.0 [5.0]		6.0 [10.0]	1.5 [4.0]	1.5 [3.0]	.75 [1.5]
Granular/pellets							1.7 [6.0]	
Mechanical								
Foliar	2.5 [4.0]	4.0 [7.0]	4.0 [6.0]	2.0 [3.0]	7.8 [12.0]	1.5 [4.0]	1.7 [6.0]	.75 [1.5]
Granular/pellets							1.7 [6.0]	
Manual Ground								
Granular/pellets							1.7 [6.0]	
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	2.0 [3.0]	2.0 [4.0]	1.0 [3.0]	2.0 [3.0]		1.0 [4.0]	0.5 [4.0]	.75 [1.5]
Basal bark/stem		1.7 [3.0]	1.2 [3.3]					
Basal soil/soil spot							1.7 [6.0]	
Cut surface (frill, injection cut stump, hack and squirt, etc.)	2.0 [4.0]			1.5 [2.3]		1.3 [4.0]		.75 [1.5]

[ ]--Maximum anticipated

[a] Values are estimated; chemical or application method not currently used.

Table 4-6 (continued)

Typical and maximum anticipated application rates in pounds per acre a.i. in Region 8

Application Method	Light Fuel Oil	Limonene	Picloram	Sulfometuron Methyl	Tebuthiuron[a]	Triclopyr Amine	Triclopyr Ester
Aerial[a]							
Foliar	0.5 [1.5]	0.9 [3.6]	0.5 [1.5]	0.13 [0.4]	1.0 [6.0]	3.0 [6.0]	4.0 [8.0]
Granular/pellets					1.0 [6.0]		
Mechanical							
Foliar	2.0 [3.5]	0.9 [3.6]	0.7 [1.4]	0.17 [0.4]	1.0 [6.0]	4.0 [8.0]	4.0 [8.0]
Granular/pellets					1.0 [6.0]		
Manual Ground							
Application method Granular/pellets							
Foliar-backpack/hand-sprayer (directed, herbaceous, etc.)	1.5 [2.5]	0.9 [1.8]	0.4 [0.5]	0.06 [0.25]	4.0 [7.0]	1.4 [2.0]	1.0 [3.0]
Basal bark/stem	1.0 [2.0]	0.9 [2.7]					1.9 [3.0]
Basal soil/soil spot					4.0 [7.0]		
Cut surface (frill, injection cut stump, hack and squirt, etc.)			0.3 [0.6]			1.0 [2.5]	

[ ]--Maximum anticipated

[a] Values are estimated; chemical or application method not currently used.

Table 4-7

Routes of exposure considered in this risk assessment

Category	Doses from Direct Exposure	Doses from Indirect Exposure
Routine		
Workers	Dermal plus inhalation dose (based on field studies)	Dermal dose from reentry to treated area based on field data
General Public	Dermal dose[a] from drift (based on modeling)	Dermal dose from reentry to treated area based on field data. Oral dose from consuming food and water with residues due to drift or inflow into stream or aquifer.
Accidental		
Spraying	Dermal dose[a] to member of public directly sprayed	Oral dose to member of public who eats directly sprayed food items
Spills	Worker dermal dose from spill of concentrate on skin	Oral dose to member of public from drinking water contaminated by an herbicid spill

[a] Inhalation is negligible based on field study data.

the area of the drifting spray droplets or where there are vapors from a volatile herbicide. However, a variety of studies have shown that inhalation exposure is very small compared with dermal exposure. In this analysis, inhalation doses have not been estimated separately for workers. They are included with dermal doses in the estimated total worker doses based on herbicide levels in the urine of workers in field experiments.

The single most important source of exposure to persons who do not handle the herbicide containers or spray equipment in routine operations is from the drift off target of airborne herbicide spray droplets. Members of the general public who are within the area of drift of the smaller spray droplets may, like workers, receive dermal and inhalation exposure. However, these exposures are relatively low compared to the exposures of workers directly involved in the spraying operations. Field studies of workers have consistently shown that inhalation exposure represents only a small part of the total exposure, so doses to the general public in this analysis have been calculated only for dermal and dietary routes (for example, Draper and Street, 1982; Libich et al., 1984).

Herbicide may be ingested from food containing herbicide residues. Food items such as garden vegetables, wild berries, or game animals may have received some level of herbicide from spray drift. Game animals may have fed on plants from the treated or drift area. Ingestion exposure could also result from drinking water that has received herbicide drift or from eating fish from a body of water that has received herbicide drift or inflow.

For routine operations, doses to workers can be significantly reduced through the use of protective clothing and equipment and adherence to proper cleanup procedures and label precautions. The amount and extent of drift from a spray site can be reduced by spraying under favorable weather conditions and using spray equipment that limits the number of smaller droplets.

In the event of an accident, workers and members of the public may be exposed to much greater amounts of herbicide than they would under normal circumstances. Workers who spill the concentrate or some of the prepared spray mixture on their skin during mixing, loading, or spraying operations or who are doused when a transfer hose breaks would be dermally exposed, as would workers or members of the public who are accidentally sprayed with herbicide because they are beneath a spray aircraft or are too close to a truck or backpack applicator.

The dermal dose would depend on the concentration of herbicide in the spray mix, absorption potential of the herbicide, the area of the sprayed person's exposed skin, the extent to which the person's clothing absorbed herbicide (some clothing is water repellent, but other material would permit penetration of the herbicide to the skin), and the time that elapses before the person can wash. Indirect dermal (reentry) exposure may occur if workers or members of the public brush up against wet vegetation in the sprayed area.

Members of the public may accidentally be exposed to the herbicide by eating food that has been directly sprayed. For example, someone could eat berries that have been directly sprayed, or they may eat meat from deer that have recently foraged on a sprayed site. Exposure to an herbicide is possible if a container of herbicide concentrate were to break open and spill into a drinking water supply, or if an aircraft were to jettison a load of herbicide into water in an emergency.

To make reasonable estimates of the possible herbicide doses to workers and the public, a number of exposure scenarios are used that represent an array of possible exposure situations. The exposure scenarios were designed to provide a range of human dose estimates, from typical to maximum, for normal operating conditions. Accident scenarios--direct application, spills on the skin, and large spills into bodies of water--are used to estimate the highest doses that could ever be expected to occur.

All but the lowest doses from all vegetation management projects conducted in Region 8 should fall within the range of doses predicted in these scenarios.

The scenarios specify those characteristics of each kind of herbicide application operation that determine human doses. For example, for workers involved in backpack operations, the number of work hours and the herbicide application rate are used to determine their doses. For aerial applications, the number and size of the sites treated in a day's operation and the herbicide application rate are used to determine doses to workers. To calculate doses to nearby residents who may eat a garden vegetable containing herbicide residue, it was necessary to estimate how much residue was on the vegetable and to specify how much of the vegetable was eaten.

The exposure scenarios are not intended to show what necessarily will happen as a result of a given treatment operation, but what could happen if all of the conditions specified in the scenario were met in the actual operations. For example, maximum worker doses are based on actual dose levels found in field exposure studies in which no protective clothing or equipment was worn. If workers wear protective clothing and equipment as required during actual operations, their doses would be significantly lower than those estimated here. However, despite all precautions, workers present during treatment operations will be directly and indirectly exposed to some herbicide.

Additional factors must be recognized when evaluating the likelihood of a member of the public receiving an herbicide dose. A forest user would receive a dose only in the immediate vicinity of the treatment area and only at the time of the herbicide application. However, because of the limited area of forest being treated and the public's use pattern, the possibility of this occurrence is slight. Likewise, a nearby resident would receive a dose as high as the one estimated in this analysis from eating garden vegetables with herbicide residue only if all of the following conditions were met:

(1) The resident's garden was close enough to a particular treatment area to receive some level of herbicide drift.

(2) The weather conditions on the day of treatment were such that the herbicide happened to drift offsite in the direction of the garden.

(3) The resident ate the vegetable immediately after the herbicide residue landed on it.

A combination of factors makes the possibility of the resident receiving such a dose highly unlikely. First, most treatment areas are located considerably further from any residence than the distance assumed in this analysis--30 to 150 m (100 to 500 feet). Second, mitigation measures described in section 2 reduce the likelihood of drift onto a garden, even if one happened to be nearby. Third, there is only a small possibility that the resident would immediately pick and eat a garden vegetable (without washing it) that had herbicide residue from that operation.

The types of representative exposures to be calculated using the methods outlined in succeeding paragraphs are listed in table 4-8. Doses were calculated for typical and maximum cases for each type of exposure listed for the public and workers, but only one dose was calculated for each type of accident. Margins of safety will be presented in section 5 for each of these exposures. The following paragraphs examine alternative assumptions for some types of exposure, but only one representative set of assumptions are used in subsequent calculations.

Table 4-8

Representative exposures

- Public
  - Dermal
    - Drift
    - Onsite-hiking, berry picking, hunting, birding, photographic trips, etc.
  - Dietary
    - Water
    - Fish
    - Meat
    - Vegetable (for example, legumes or salad plants)
    - Berries

- Workers
  - Aerial
    - Pilot
    - Mixer/loader
    - Observer
  - Mechanical Ground
    - Applicator
    - Mixer/loader
    - Applicator/mixer/loader
  - Manual Ground
    - Backpack
    - Soil spot
    - Basal stem
    - Cut surface

- Accidents
  - Spill onto worker
  - Accidental spray
  - Spill into water
    - Ground--5 gallons of concentrate into a pond
    - Air--100 gallons of spray mix into reservoir

Public Exposure and Dose Estimation

Field studies of actual herbicide doses to the public under application conditions typical of those in Region 8 are not available. Consequently, it was necessary to estimate public doses by modeling the transport and fate of the applied herbicides. Field studies conducted under the most nearly equivalent conditions were used whenever possible as a basis and a check for the models.

Spray Drift

The potential for herbicide sprays to drift onto adjacent lands or into nearby bodies of water was assessed based entirely on the results of empirical studies reported in the scientific literature. The analysis considered deposition on surfaces, including exposed skin, as well as water, game animals, and various classes of plants that may contribute directly or indirectly to the human diet.

Specific field studies were chosen to represent equipment and conditions that most nearly represent those expected for aerial and ground broadcast spray applications. The Forest Service intends to use spray equipment that produces uniformly large droplets with very few fine droplets. Fine droplets (less than approximately 100 microns) are normally responsible for offsite drift. Low-drift nozzles that may be used for aerial applications include the TVB, Microfoil, and Raindrop nozzles. Ground-based mechanical spray systems typically use Raindrop or similar nozzles that produce large droplets. Wind-tunnel simulations (Yates, Cowden, and Akesson, 1985) have demonstrated the large droplet sizes produced by Raindrop nozzles. For example, the median droplet diameter (by volume) from an RD-10 Raindrop nozzle ranged from 1170 to 1460 microns, when it was aimed with the air stream. For the RD-7 Raindrop nozzle, the median diameter ranged from 960 to 1080 microns. Less than 1 percent of the spray volume was in droplets less than 154 microns for both nozzles. Field measurements of spray drift have also demonstrated the low drift potential of Raindrop, TVB, and Microfoil spray systems (Yates et al. 1978; Fears and McMaster, undated).

Drift of sprays applied by aircraft has been estimated for this risk assessment based on data presented by Yates et al. (1978) of the University of California at Davis. Mylar fallout sheets were used to measure glyphosate spray deposition at various points downwind of a Bell 47G-5 helicopter using a Microfoil boom. The test was performed in an open field under stable atmospheric conditions with winds averaging 2.6 mph. Drift of sprays applied by ground equipment was estimated based on a field test reported in Yates et al. (1978). In this test, glyphosate was applied when winds were 8.5 mph by a ground sprayer with an 8003 fan nozzle. This spray system is expected to cause greater drift than spray systems typically employed in Region 8, but field measurements of drift are unavailable for the spray equipment typically used by the Forest Service and its contractors (e.g., 8004LP low pressure, 2503, and OC40 nozzles). To illustrate the range of drift measurements that have been observed, data will also be presented from a second ground sprayer test, using a low-pressure KGF10 sprayer. During this test, atmospheric conditions were stable and the wind averaged 5.1 mph.

To facilitate use of the data from the various published field tests discussed above, a computer program was written to show how residues accumulate from multiple swaths (the long, narrow pattern of herbicide laid down by a broadcast sprayer such as a helicopter) and to correct for various application rates and swath widths. The program was then run to calculate deposition at selected representative distances for a nominal application rate of 1.12 kg/ha (1 lb/ac). The results are shown in figure 4-1 for the aerial spray system. The deposition at 25 m is 0.15 g/ha, assuming that 1.12 kg/ha is applied to a 16.2-ha (40-ac) spray block. The deposition declines rapidly with distance. At 100 m, the deposition is only 0.006 g/ha. The results for ground-based spray systems are shown in figure 4-2. Again assuming a 16.2-ha spray block, the deposition for the KGF10 sprayer is 0.09 g/ha, and it declines to about 0.07 g/ha at 75m. The deposition from the 8003 nozzles is considerably greater, with 3.9 g/ha at 20 m, and it declines to 1.8 g/ha at 75 m. This case was used to estimate representative exposures downwind of all silvicultural projects because most broadcast applications are expected to be done with ground-based equipment, and this case is based on data that deliberately represent relatively high-drift (but still realistic) conditions. This approach is deliberately conservative, in that it is intended to avoid any possible underestimation of exposure due to drifting herbicide. Exposures calculated based on aerial application with low-drift nozzles would not be greater than those calculated here for a ground-based spray system.

Downwind drift of spray from right-of-way applications is expected to be less than from silvicultural applications because the area to be sprayed is relatively narrow. To demonstrate this effect, spray drift calculations were again done based on the Microfoil test by Yates et al. (1978). This test can be used to estimate drift downwind of right-of-way applications because measurements were taken downwind of a single spray swath. The right-of-way has been assumed to be 20 m wide. The results are shown in figure 4-3 for an application rate of 1.12 kg/ha assuming that the wind is perpendicular to the right-of-way. The calculations show that the drift downwind from a right-of-way application is less than from a larger spray block, but the difference decreases somewhat with distance. At 20 m, the deposition downwind of a right-of-way is about 60 percent as great as downwind of a 40-ac spray block; and at 75 m, it is about 71 percent.

Similar calculations based on the ground sprayer test demonstrated that right-of-way applications will have significantly less drift compared with silvicultural applications. At 20 m downwind of a right-of-way application, the deposition is expected to be 16 percent as great as downwind of a 16.2 ha block. At 75 m, the ratio is 31 percent.

During calculation of representative exposures, a 16.2-ha spray block (figures 4-1 and 4-2) was assumed for all herbicides that are expected to be used for silvicultural applications, even though they may be used for rights-of-way also. If an herbicide is used only for rights-of-way, then the drift estimates were corrected using the ratios discussed in the preceding paragraph.

Residues on Plants

Herbicide residues on plants on treated sites were estimated based on factors reported by Hoerger and Kenaga (1972). These factors were derived from a large number of studies, and they allow prediction of residues in parts per million (ppm) based on the application rate in pounds per acre. These residue estimates were calculated assuming no herbicide degradation, so they apply to conditions immediately after application. Following

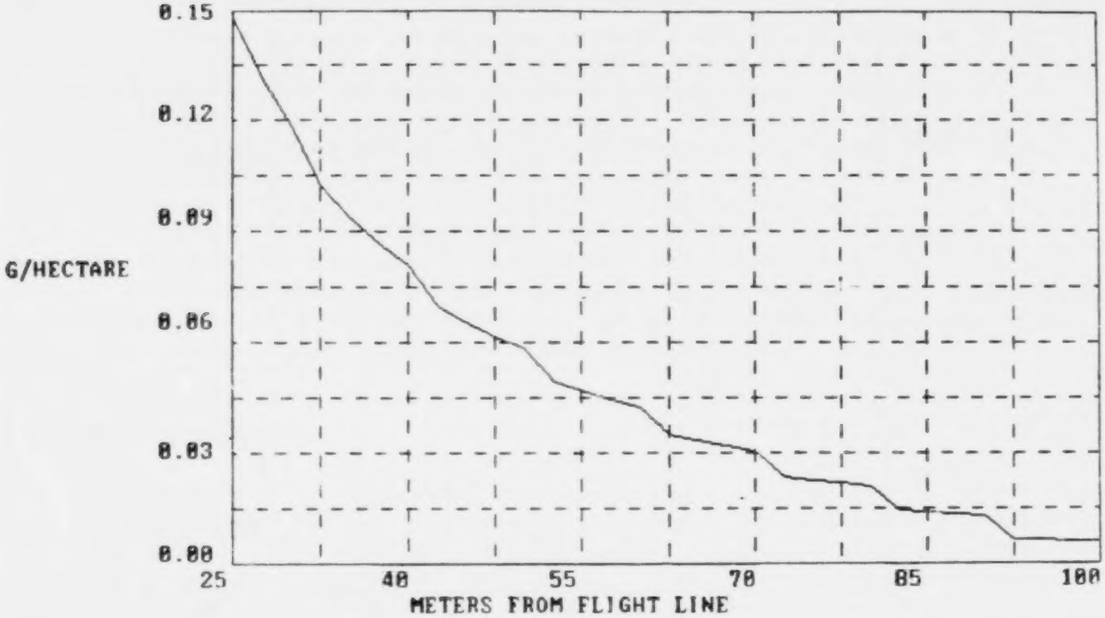

Figure 4-1--Spray deposition downwind of a 16.2 ha spray block--aerial application of 1.12 kg/ha with a microfoil boom

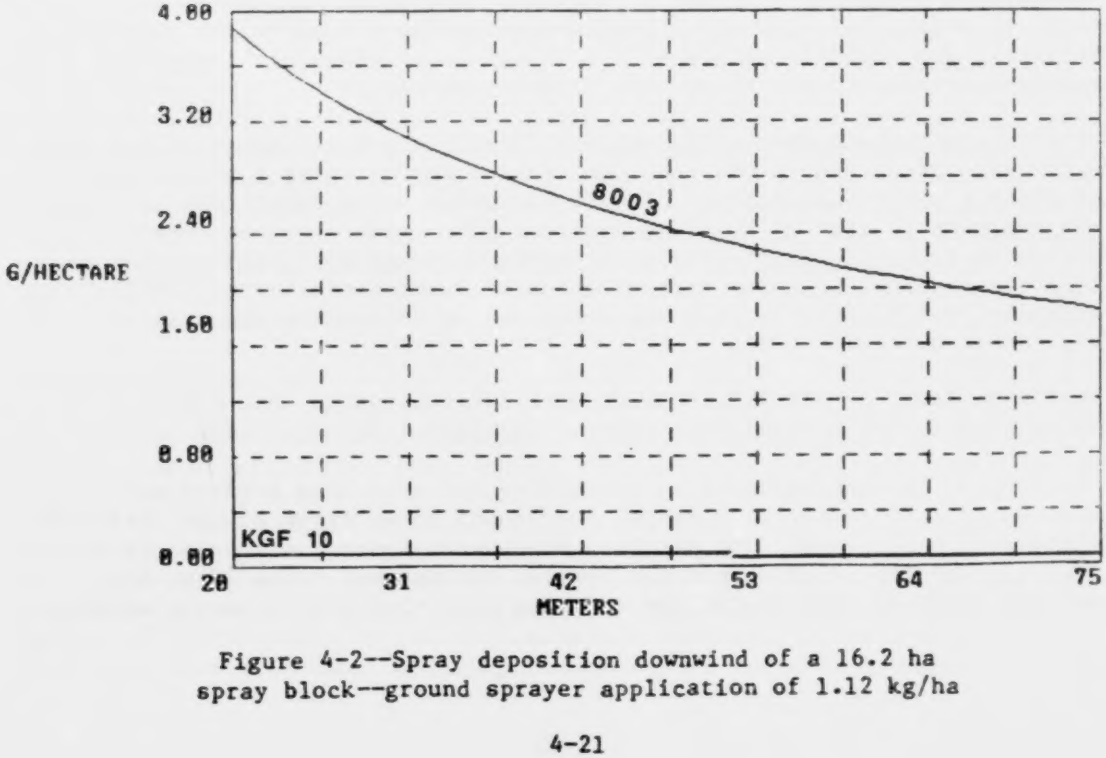

Figure 4-2--Spray deposition downwind of a 16.2 ha spray block--ground sprayer application of 1.12 kg/ha

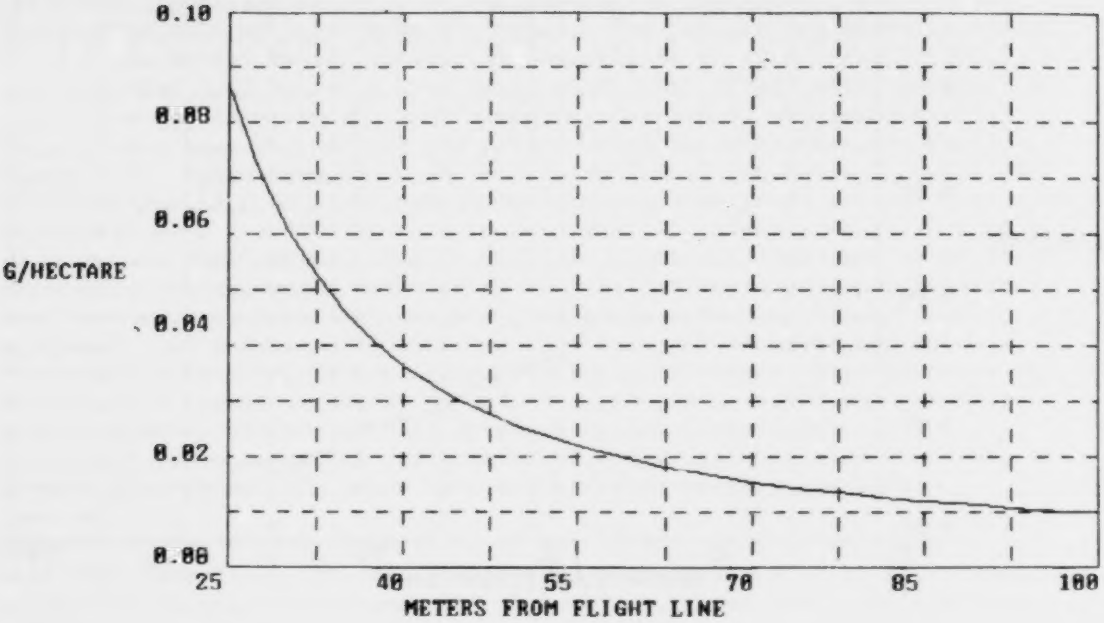

Figure 4-3--Spray deposition downwind of a right-of-way--aerial application at 1.12 kg/ha with a microfoil boom

Hoerger and Kenaga (1972), the plants were classified into broad groups based on vegetative yield, surface-to-mass ratio, and plant interception factors. The residues estimated for each type of plant are intended to represent realistic yet relatively high estimates.

Offsite plant residues were calculated first for grasses based on the spray drift data discussed in the previous section and by using a regression equation given in Yates et al. (1978) to relate spray deposition on young wheat plants to that on sampling devices. The deposition was then estimated for other plant groups, including berries and leafy vegetables, by using the same relative factors given by Hoerger and Kenaga (1972), assuming that deposition on young wheat was approximately the same as deposition on range grass.

Herbicide doses to individuals were calculated assuming that they eat 400 g (0.9 lb) of contaminated berries or legumes (peas or beans).

The typical minimum distance to residences and vegetable gardens was assumed to be 61 m for both aerial and ground spray applications, based on historical experience. The absolute minimum distance was assumed to be 30.5 m. The typical distance for berries was assumed to be 20 m, but maximum residues were calculated assuming that they were directly sprayed.

Residues in Water Resulting From Spray Drift

Residues in drinking water were calculated assuming that the water is 0.305 m deep and that the herbicide spray drifts directly downwind to the water body over a specified buffer distance. The typical buffer distance was assumed to be 20 m, and the minimum distance was assumed to be 10 m. The actual residues in water would be less under more favorable spray conditions, at greater distances, or with larger water bodies. Dilution or degradation would decrease residues. Herbicide doses to individuals were calculated assuming that they drink 1 liter of the maximally contaminated water. Fish were assumed to come from water 0.61 m deep. Many water bodies, especially ponds and lakes, are deeper than those considered here, but these deeper water bodies would have proportionately lower concentrations.

Residues in Game Animals

Residues were calculated for one representative game animal: a 60-kg deer. The entire body surface area of the animal was assumed to be exposed to spray drift. This is likely to overestimate exposure because only a portion of the body surface is exposed to drift at any one time. Forty percent of the body surface was assumed to come into contact with vegetation and thereby gain an additional average dermal residue level equal to that on the vegetation. Penetration of the herbicides through animal skin was assumed to be the same as through human skin. This assumption also may overestimate doses, for example, by ignoring the protective effect of fur.

The deer were assumed to get an oral dose both by grooming and in their diet. The dose from grooming was assumed to amount to 29 percent of the nonabsorbed dermal dose. The deer diet was assumed to consist of 2.45 kg of forage plants and 4 liters of water per day, both containing herbicide.

The concentration of herbicide in game meat was calculated by summing the animal's doses from both the dermal and oral routes of exposure and by assuming that 10 percent of that total dose was retained in the meat of the animal. This is similar to the method used in the exposure analysis of USDA (1984). This degree of retention assumes that the deer is killed soon after exposure because none of the herbicides are known to accumulate in meat. Herbicide doses to humans were calculated by assuming that they eat 400 g of deer meat in 1 day.

Residues in Fish

Residues in fish were calculated assuming that the fish lived in and were caught from waters 0.61 m deep, directly downwind of a treated site, with a typical buffer distance of 20 m and a minimum buffer distance of 10 m. For most of the herbicides considered in this analysis, which do not appreciably bioaccumulate, the concentrations in fish were taken to be equal to the particular herbicide's concentrations in water. For one herbicide for which bioconcentration is likely to be greater--tebuthiuron--a bioconcentration factor of 10 was used. Doses to humans from eating fish containing herbicide were calculated assuming that 400 g are eaten in 1 day.

Dermal Exposure of Forest Users

Dermal exposure resulting from drift was estimated by assuming that 0.186 m^2 of skin were exposed and the level of deposition on skin is the same as that found on the sampling sheets used in the drift monitoring studies. Drift distances were assumed to be the same as discussed previously for residences. The dose was calculated as the deposited amount times the dermal penetration rate.

Dermal absorption values have been reported for some of the herbicides evaluated in this risk assessment, ranging from 0.1 percent for 2,4-DP to 6.9 percent for dicamba (based on Makary et al., 1986). The dermal absorption of 2,4-D is about 6 percent (Feldman and Maibach, 1974), and the absorption of picloram has been reported to be 0.18 percent (Nolan et al. 1983, as cited in Mullison, 1985). The dermal absorption of triclopyr is 1.65 percent (Dow, 1987c). Dermal absorption estimates were not available for the other herbicides or adjuvants considered in this risk assessment. However, dermal absorption values for several other herbicides were all reported to be less than 10 percent (Grissom et al., 1985; Wester and Maibach, 1985; Yi-lan et al., 1984; and others). Therefore, 10 percent dermal penetration was used as a conservative estimate for the herbicides on which no dermal values were available. The dermal penetration of light fuel oil is also unknown, but it has been conservatively estimated to be 25 percent. Several factors may affect the dermal absorption of herbicides, such as the dosage applied, the solubility of the chemical, the variation between species, and external conditions (temperature and humidity) (Yi-lan et al., 1984). However, the most important factors by which the amount absorbed can be decreased are the use of protective clothing and washing after skin contamination (Yi-lan et al., 1984; Wester and Maibach, 1985). The longer the pesticide remains on the skin, the greater the percent absorbed. For example, the rate of absorption of dicamba is estimated as 0.29 percent per hour (Makary et al., 1986). Because this rate of absorption is considered low, washing would effectively lessen exposure time and therefore decrease the amount of the dose absorbed. With these protective measures in mind, percent dermal absorption would be expected to be less in all cases than that estimated in this risk assessment.

Dermal doses from incidental contact with foliage, represented by vegetation contact for the hiker, were estimated based on a field study. Lavy et al. (1980) measured the level of a phenoxy herbicide on cloth patch samplers attached to a person who walked through a treated forest area. The residues were less than the detection limit of 0.01 mg/100 cm^2 patch, but in this analysis a conservative assumption was made that the residues were at the detection limit. The area of clothing contacting foliage was assumed to be 40 percent of the total human surface area, and 10 percent of the total area was assumed to be bare skin contacting foliage. The same dermal penetration rates discussed previously were applied to bare skin, but the penetration through clothing was assumed to be 30 percent over a 6-hour period, based on work by Newton and Norris (1981).

The Potential for Herbicides To Contaminate Ground Water

For ground-water contamination by herbicides to be a problem, the herbicides must be carried, by percolating water, into an aquifer. They must appear in the aquifer at concentrations sufficient to cause a potential health hazard, and the water must be used for human consumption. Another possible type of problem can arise if phytotoxic concentrations occur in the aquifer and the water is used for irrigation.

Movement of herbicide to and through an aquifer can be broken into several stages:

(1) Movement into the soil.

(2) Movement through the rooting zone.

(3) Movement from the rooting zone into the aquifer.

(4) Movement within the aquifer to a point of water use.

Movement into the Soil

The amount of herbicide moving into soil from the soil surface or through plants depends on a variety of factors, including the amount applied, method of application, soil characteristics, climate, hydrology, and properties of the herbicide. The amount applied in forestry is typically in the range of 0.2 to 9 kg/ha, but only 1 to 3 times in the life of a stand (25 to 100 years for pines; 60 to 200 years for hardwoods). Relevant properties of the herbicide at this stage include its tendency to volatilize or degrade, for example, by photolysis, microbial activity, or hydrolysis. Most forestry-use herbicides are applied internally or to plant surfaces, so their interaction with the plant is important. Because herbicides are intended to kill plants, they generally have the ability to penetrate plant surfaces. Some of the penetrating herbicides remain in the plant tissues close to the site of application, while others, such as 2,4-D, can be freely translocated within the plants. If translocation is into the roots, some can be a source of herbicide movement into the soil; others, like glyphosate, adsorb to soil and do not move beyond the immediate zone of root contact. Release from roots can occur either through exudation by living root tissue, or during decomposition of the root tissue after plant death. Other herbicides, such as hexazinone, are soil-active so that they can be applied in a manner that promotes their penetration into the soil, where they contact plant roots. Once within the plant, the herbicide may or may not be degraded by plant metabolism. The metabolism of herbicides by plants, as well as other environmental fate characteristics, have been summarized in USDA Handbook No. 633 (1984). Interaction of the herbicides with plants will not be described in detail here, except to point out that a significant fraction of the applied herbicide in many cases never enters the soil. At the other extreme, essentially all of the herbicide can contact the soil if it is soil-applied, or if a rainstorm occurs soon after application. This extreme assumption of 100 percent soil contact is used in leaching calculations presented below. Early formulations of 2,4-D were relatively volatile, with 50 percent sometimes lost to the atmosphere. However, formulations in current use are amines or esters, which are designed to reduce volatility.

Movement Through the Rooting Zone

The second stage of movement is through the rooting zone of plants. This may be the most complicated stage of movement because the herbicide interacts with all soil components (minerals, organic matter, microorganisms) as well as with plants. Important surface hydrologic factors that influence herbicide movement include rainfall, interception, percolation, runoff, and evapotranspiration. Net movement through soil depends on the relative rates and routes of water seepage, adsorption, degradation, and dilution. EPA (Carsel et al., 1984) has sponsored the development of a Pesticide Root Zone Model (PRZM) intended to simulate these factors in an integrated manner. The PRZM model is data- and computer-time-intensive, so the Leaching Evaluation of Agricultural Chemicals (LEACH) methodology (Dean et al., 1984) was developed to allow identification of leaching-frequency curves based on degradation rate, adsorption (expressed as a simple partition coefficient), climatic factors, and soil characteristics. Each leaching-frequency curve is based on a 25-year simulation using PRZM with local rainfall records and typical crop characteristics. The areas considered by the LEACH handbook include major agricultural areas of the United States, which coincide partially with major areas of forest herbicide use. However, the crops considered by the handbook are restricted to row crops and grains. The LEACH methodology is still useful for illustrating the generally low potential for forestry herbicides to leach below the rooting zone.

Soil textural class--sand, silt, clay, and various combinations--influences the potential for leaching in several ways. Sand contains relatively large particles and therefore has less surface area than the same volume of clay, and silt is intermediate. Therefore, adsorption is typically greater to clay than to sand. The fraction of pore space and hydraulic conductivity are typically greater for sand than clay.

The LEACH methodology was applied to herbicides that represent a range of mobility and persistence, assuming average characteristics for a sandy loam soil, which has a moderately high leaching potential. A corn-growing area of the southeastern coastal plain, "site no. 13," was used to provide an example with moderately high rainfall (127 to 152 cm annually) and a rooting depth of 90 cm. SCS runoff curve number 77 was assumed, indicating moderately high runoff potential.

The results of the LEACH analysis are presented in table 4-9 as the fraction expected to leach below the rooting zone 10 percent of the time. Ninety percent of the time, the fraction leaching would be less than that shown in the table. The fraction leaching for chemicals with low to moderate leaching potential, such as sulfometuron methyl, is essentially zero. Even those chemicals with a relatively high leaching potential have only a small probability of leaching beyond the root zone. For example, the fraction of hexazinone expected to leach beyond the root zone 10 percent of the time is no more than 5 percent. Among the most mobile of the herbicides are dicamba and tebuthiuron, for which the fraction leaching beyond the rooting zone 10 percent of the time is 12 and 9.5 percent of the applied amount, respectively. The LEACH methodology predicts that under these conditions most herbicides will not leach significantly below the

Table 4-9

Leaching potential of selected herbicides in sandy loam or similar soil type

Herbicide	Adsorption Coefficient[a] (K_d)	Retardation Factor (R)[y]	Half-life[a] (days)	Fraction Leaching[b]
2,4-D	0.49[c]	4.5	<28[d]	NS[e]
2,4-DP	0.49[f]	4.5	10[g]	NS
Dicamba	0.11[h]	1.8	25[i]	12.0
Fosamine	20.00[j]	145.0	<10[k]	NS
Glyphosate	16.50[l]	119.8	61[m]	NS
Hexazinone	0.20[n]	2.4	<30[k]	5.0
Imazapyr	0.28[o]	3.0	27[p]	4.7
Light fuel oil	0.83[q]	7.0	6[q]	NS
Picloram	0.63[h]	5.5	63[r]	2.0
Sulfometuron methyl	0.71[s]	6.1	10[t]	NS
Tebuthiuron	2.40[u]	18.3	392[v]	9.5
Triclopyr	1.50[w]	11.8	46	NS

[a] K_d was determined for sandy loams in most cases; when values were not available for sandy loam, silty clay values were used. Half-life was determined for actual degradation in some cases, but many of the values were estimated based on field dissipation. See individual references for details.

[b] Estimated from Leaching Evaluation of Agricultural Chemicals (LEACH) Handbook prepared for EPA by Dean et al., 1984. Fraction (of the applied chemical) leaching is for 10 percent of the time, i.e., 90 percent of the time the fraction would be less than that indicated. Fractions could not be calculated for chemicals where a K_d or half-life was not available.

[c] Average value for 7 soils, D-18.

[d] Stewart and Gaul, 1977.

[e] NS = not significant.

[f] Used 2,4-D value, no information on 2,4-DP.

[g] Alton and Stritzke, 1973.

[h] Average from Rao and Davidson (1980) in LEACH Handbook.

[i] 17 to 32 days in Alton and Stritzke, 1973 in USDA, 1984.

[j] Silt loam soil, DuPont, 1975 in Ghassemi et al., 1981.

[k] Based on average literature values, in Neary, 1985.

[l] Sprankle et al., 1975.

[m] Average of 11 soils in USDA, 1984.

[n] Rhodes, 1980.

[o] Calculated based on equation 4 from Lyman in LEACH Handbook, assuming 1% OC.

[p] Michael, J.L., 1986.

[q] From draft superfund Public Health Evaluation Manual. Half-life assumed to be same as for surface waters, 1% OC assumed.

[r] Average from Nash (1980) in LEACH Handbook, calculated from K_s.

[s] Harvey et al., 1985.

[t] Average from Michael and Neary, undated.

[u] Chang, 1965 and Chang and Stritzke, 1977 both in USDA, 1986.

[v] Elanco Products Company, 1983 in USDA, 1986.

[w] Marks lower 10% of range of observed K_d's.

[x] Ghassemi et al., 1981.

[y] R is the ratio between the velocity of the solvent (in this case, water) and the velocity of the contaminant.

rooting zone. The relatively mobile and persistent herbicides can be expected to leach partially out of the rooting zone when rainfall conditions are conducive, but even in these cases a substantial portion of the herbicide does not leave the rooting zone.

The LEACH methodology can be used to estimate the concentration of herbicide at the bottom of the rooting zone as a starting point for estimation of the subsequent leaching from the bottom of the rooting zone down to the aquifer at the depth of interest. The LEACH methodology as applied above only considered the surface 90 cm of soil. Aquifers used as practical water sources are typically tens or hundreds of meters deeper. The herbicide will be subject to further adsorption, degradation, and dispersion as it travels this distance.

Leaching Under Extreme Conditions

A second model was used to investigate the potential for leaching into ground water under conditions unusually conducive to leaching. Heavy rainfall is assumed to occur soon after application, and 100 percent of the herbicide is assumed to reach the soil surface either by direct application or through foliar washoff. Degradation is assumed to be negligible over this time period, which may be 1 to 3 days after application.

The model used for this situation is a simple one-dimensional mathematical formulation. It can be used as an independent check on the reasonableness of the conclusions based on the LEACH methodology and to determine herbicide concentration profiles as a starting point for the analysis of surface runoff.

The leaching simulation provided graphs of total herbicide concentration versus depth and tables of fractions of the herbicides in each centimeter increment of soil.

The leaching model requires data describing soil and hydrologic properties. Calculations were performed assuming a sandy loam to provide a basic case that was realistic but represents moderately high leaching potential. Several leaching calculations were also done for sand to show nearly maximal leaching conditions.

The principal chemical property considered by the model is the adsorption coefficient. A linear adsorption isotherm is assumed, indicating that the concentration adsorbed to soil is a constant times the concentration in water. The model is intended to represent freely reversible equilibrium adsorption, which is a good approximation for most of the herbicides. However, glyphosate does not meet this condition very well. Consequently, for glyphosate, the model was used only to calculate concentrations at early time periods when adsorption predominates and desorption is unimportant. This presented no problem for the analysis because leaching of this chemical has been shown by field and laboratory studies to be minimal. But the leaching model still did provide an upper bound estimate of the distribution of the herbicide below the soil surface.

The leaching model is based on the one-dimensional form of the differential equation governing convective-dispersive solute transport (Travis, 1978):

$$\frac{\partial C}{\partial t} = D \frac{\partial^2 C}{\partial X^2} - V \frac{\partial C}{\partial X} - \frac{\rho}{\Theta} \frac{\partial S}{\partial t}$$

where:

C = the concentration of the solute in the soil solution

S = the concentration in the soil matrix

X = the depth into the soil

V = the X component of the Darcy soil water flow velocity

D = the solute dispersion coefficient

ρ = the bulk density of the soil

Θ = the volumetric soil water content

t = time

An assumption was made of constant coefficients in the equation. The linear adsorption isotherm used to describe the balance between chemical in the soil solution and that on the soil matrix can be expressed as follows:

$$S = K_d C$$

where K_d is the adsorption (distribution) coefficient. This allows a simplification of the convective-dispersive solute transport equation to:

$$\frac{\partial C}{\partial t} = D_o \frac{\partial^2 C}{\partial X^2} - V_o \frac{\partial C}{\partial X}$$

where:

$$D_o = D \,/\, 1 + \left[\frac{\rho \cdot K_d}{\Theta}\right]$$

and

$$V_o = V \,/\, 1 + \left[\frac{\rho \cdot K_d}{\Theta}\right]$$

Various analytical solutions to this equation have been presented by Travis (1978). The solution corresponding to an instantaneous release of a finite quantity of material M (in grams per square centimeters (g/cm^2)) is given by:

$$C(x, t) = \frac{M}{\sqrt{4\pi D_o t}} \exp - \left[\frac{(X - V_o t)^2}{4 D_o t} \right]$$

This equation was used to approximate the movement of the herbicides through soil profiles. A computer program was written to solve the transport equation and to integrate it over 1-cm segments. The output of the program includes a table of herbicide content for each centimeter of soil expressed as a fraction of the total for each time selected.

Leaching Profile Predictions. Typical parameters were input to the model for sand and sandy loam soil types. The assumed parameters were as follows:

(1) $\rho = 1.5$ g/cm^3 for sand, and 1.49 g/cm^3 for sandy loam (based on information in Brady (1974) and Carsel et al. (1984)).

(2) $\Theta = 0.437$ cm^3/cm^3 (water volume/total soil volume) for sand, and 0.453 cm^3/cm^3 for sandy loam (saturation was assumed) (based on information in Carsel et al. (1984)).

(3) D = 15 cm^2/hr for sand, and 8 cm^2/hr for sandy loam.

The dispersion coefficient is higher in the sand than in the sandy loam, and the sand has more pore space.

Several leaching profiles are presented here for two example herbicides, glyphosate and 2,4-D, to show the effect of varying adsorption, water percolation, and soil type. A single leaching profile will be presented for each of the other herbicides under a standard set of conditions.

For glyphosate, a wide range of adsorption coefficients (K_d) has been reported for various soils. Hance (1976) found an average K_d of 106 in 9 soils, with a range of 18 to 377. Spramble et al. (1975) reported a K_d of 16.5. Figure 4-4 shows the predicted profile of glyphosate applied to a sandy loam soil at 1.12 kg/ha, assuming K_d is 106. After 6 cm of percolated water, 94 percent of the glyphosate is still in the top 1 cm of soil. Figure 4-4 also shows the predicted concentration profile at the lowest observed K_d, 16.5. Even in this case, about 87 percent of the glyphosate is still in the top 2 cm of soil after 6 cm of water have percolated. The greatest leaching for glyphosate would be the case of a low adsorption coefficient combined with the sandy soil. This is shown by the curve with the lowest peak in figure 4-4, which shows that little glyphosate (less than 0.1 ppm) penetrates to 5 cm. When it is additionally considered that the half-life of glyphosate in soil is about 2 months (USDA, 1984), there is no realistic potential for contamination of ground water.

Among the forestry herbicides, 2,4-D has a relatively high mobility because of its low adsorption. Grover (1973) studied adsorption of 2,4-D to seven soils and found a range for K_d of 0.09 to 1.3, with an average of 0.49. In a study of 2,4-D adsorption to nine soils, Rao and Davidson (1980) found an average K_d of 0.78. Figure 4-5 shows the leaching profiles predicted

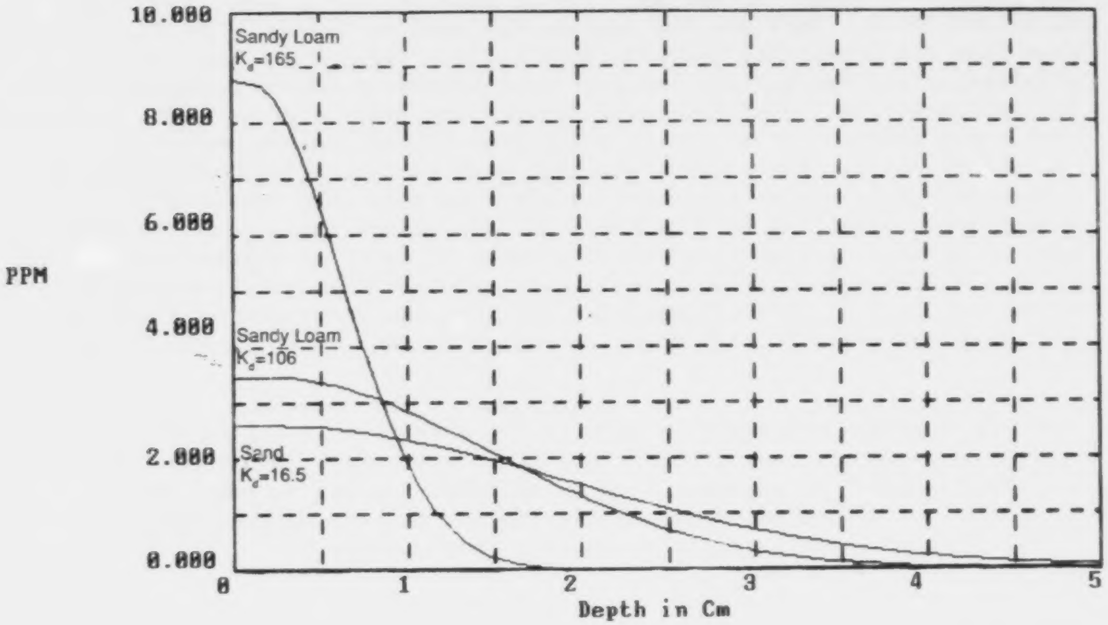

Figure 4-4--Leaching profiles of glyphosate after 6 cm of percolated water

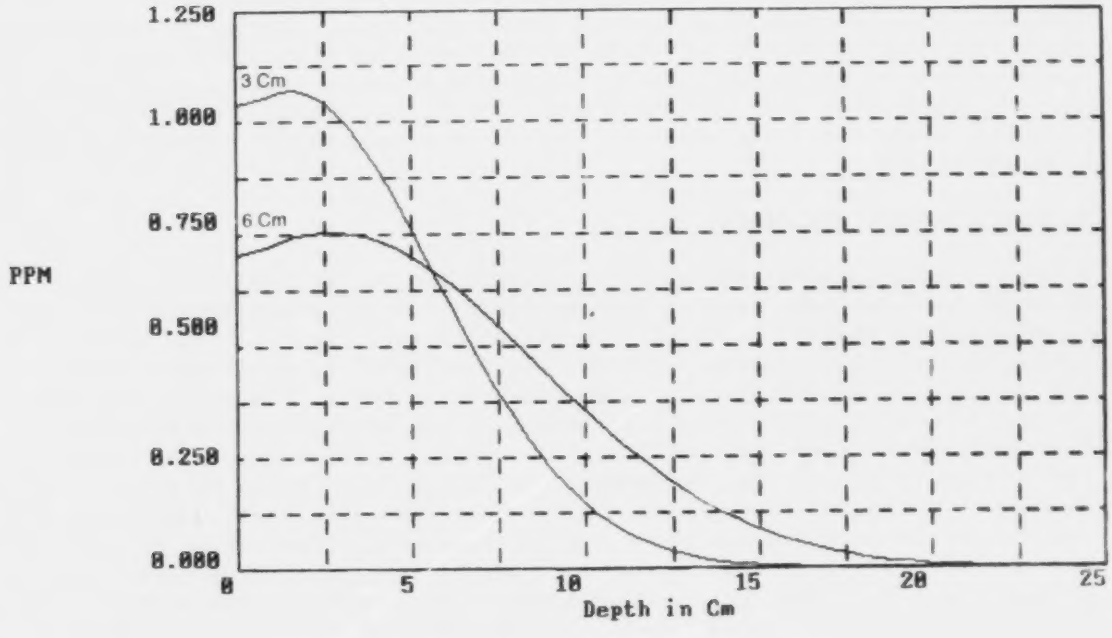

Figure 4-5--Leaching profiles of 2,4-D in sandy loam soil after 3 to 6 cm of percolated water by the model for 1.12 kg/ha of 2,4-D in a sandy loam soil with K_d = 0.49, and figure 4-6 shows the maximal rate of leaching for a sand, assuming a K_d of only 0.09. This was the minimum K_d found by Grover. The figures show that 2,4-D leaching proceeds more rapidly than in the case of glyphosate, and the surface layer of soil begins to become depleted after a few centimeters of water have percolated. The profile shows a central peak that moves downward at a rate directly proportional to the amount of percolated water. The model shows that peak concentrations will diminish appreciably before they can reach aquifers, simply as a result of dispersion. Depending on local soil and vegetation conditions, 24 cm or more of rainfall may be required to produce 12 cm of percolated water. It is very unlikely that substantially more rainfall than this would occur before residues are largely degraded. Half-lives reported for 2,4-D in soil vary considerably, but they are generally less than 1 month (USDA, 1984).

Leaching profiles are shown in figures 4-7 through 4-16 for each of the other herbicides in a sandy loam. In each case, the application rate is 1.12 kg/ha, and 6 cm of water have percolated. It can be seen that the herbicide concentrations near the surface are at most about 1 ppm, and at 25 cm, herbicide concentrations are less than 0.1 ppm.

This simulation is quite crude in several respects. The distance moved by herbicides in the field could be expected to vary with degree of saturation. The simulation has not accounted for any form of degradation. In spite of these simplifications, the simulation clearly shows that herbicide concentrations will be very much reduced before reaching any typical aquifer. Wells are very uncommon in actively managed forests, so treated areas will very rarely lie directly over a well. Wells occur more typically at a distance from treated areas, therefore, further dilution and degradation during lateral movement of the herbicide plume is likely to occur. Based on the simple simulation presented here, which agrees in its general conclusions with the LEACH methodology, concentrations of herbicide at wells in the immediate vicinity of treated forest sites are not expected to be detectable.

Movement Through The Aquifer

A two-dimensional model was used to investigate the horizontal dispersion of herbicide that may reach an aquifer under conditions deliberately chosen to show nearly maximum leaching to a potential water source. The model (using the method of characteristics) was developed and programmed by the U.S. Geological Survey (Konikow and Bredehoeft, 1986). The model computes changes in concentration over time caused by the processes of convective transport, hydrodynamic dispersion, and mixing (or dilution). The model was applied to a case that represents conditions very favorable for movement of herbicides in ground water. This case simulates conditions typical of northern Florida where the water table is often close to the surface and soils typically are sands. Runoff is minimal from these very permeable soils. Only the unconfined near-surface aquifer was modeled. In fact, this aquifer is rarely used. In some parts of Florida, this aquifer is underlain by a confining layer (the Hawthorne formation). Below the confining layer are limestones that comprise the Floridan aquifer. This

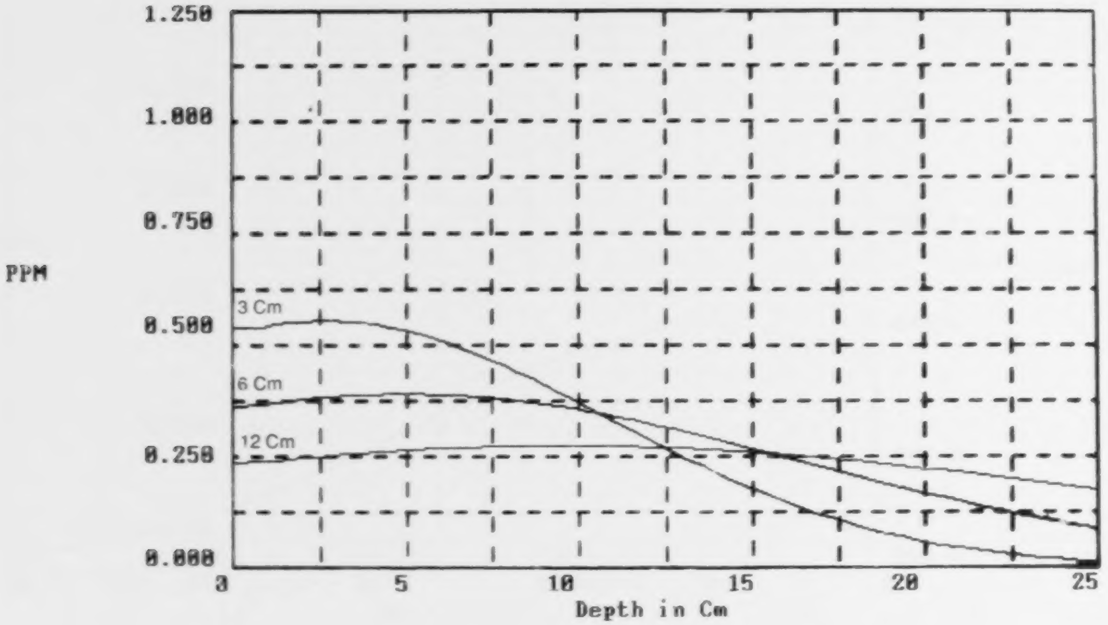

Figure 4-6--Leaching profiles of 2,4-D in sand after 3 to 12 centimeters of percolated water

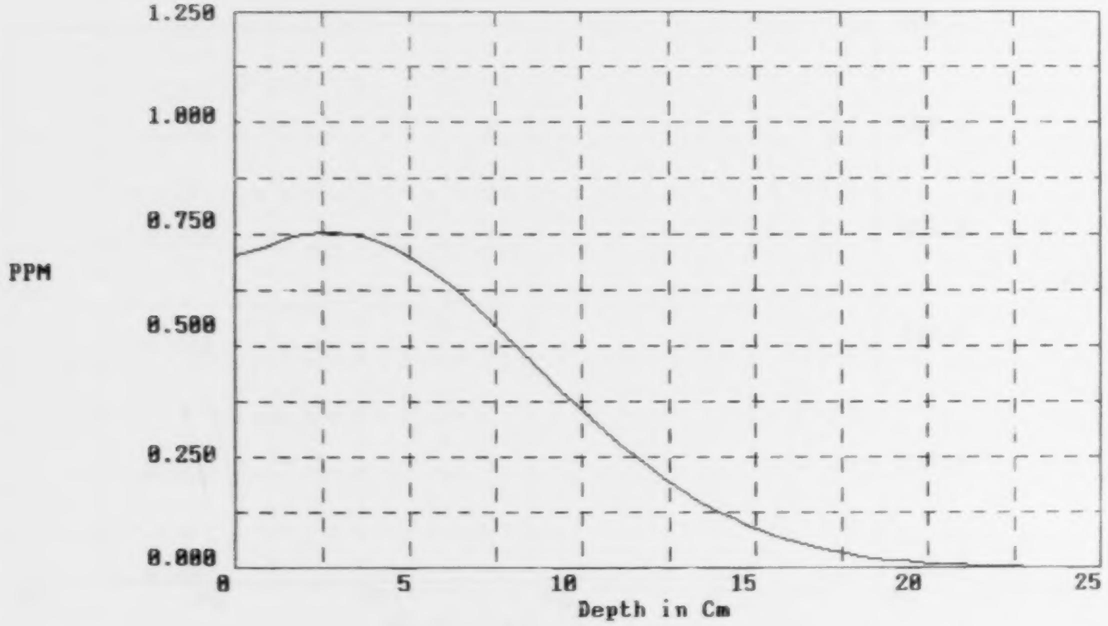

Figure 4-7--Leaching profiles of 2,4-DP in sandy loam soil after 6 cm of percolated water

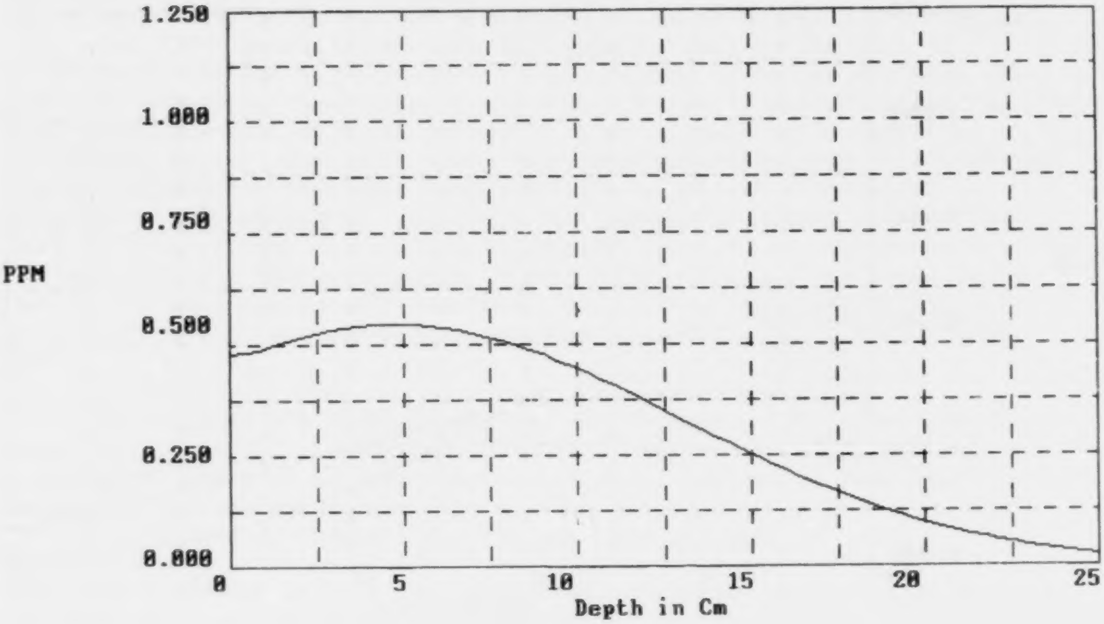

Figure 4-8--Leaching profiles of dicamba in sandy loam soil after 6 cm of percolated water

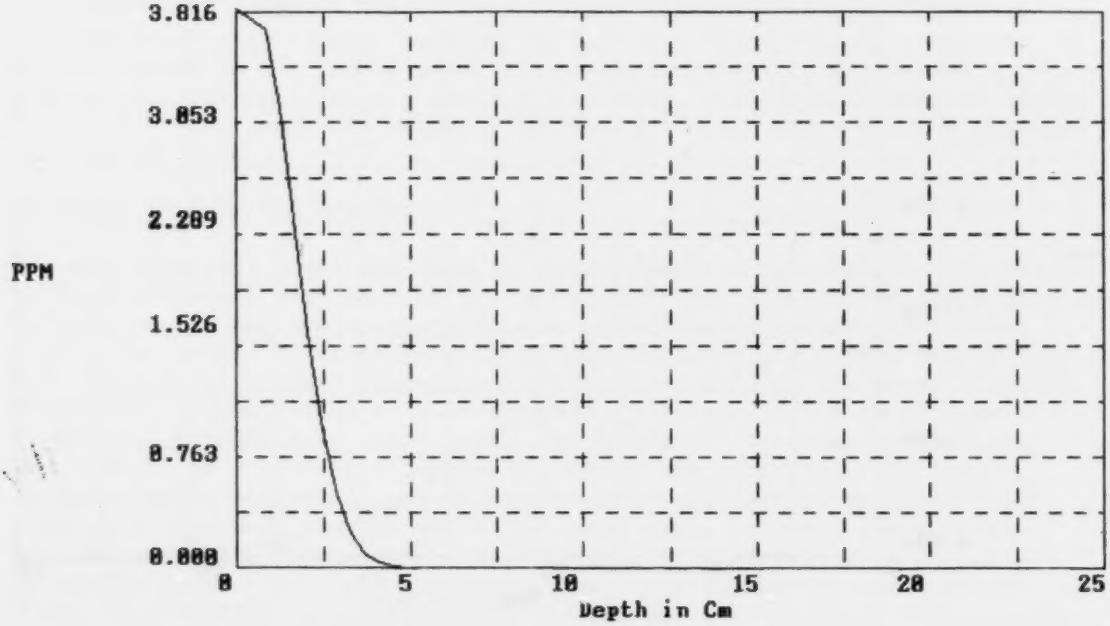

Figure 4-9--Leaching profiles of fosamine in sandy loam soil after 6 cm of percolated water

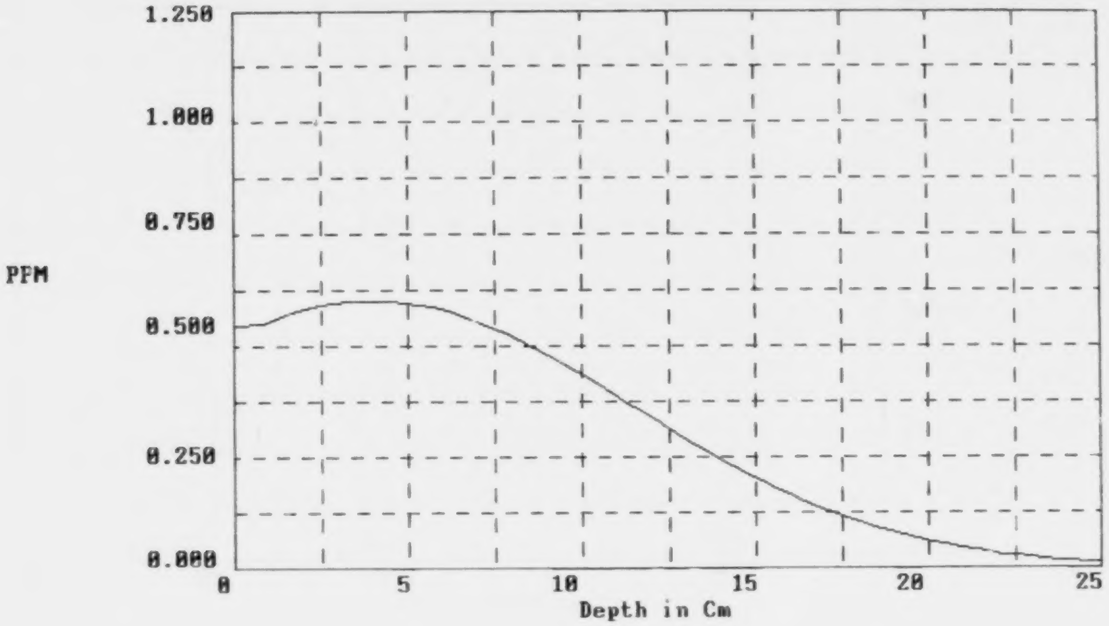

Figure 4-10--Leaching profiles of hexazinone in sandy loam soil after 6 cm of percolated water

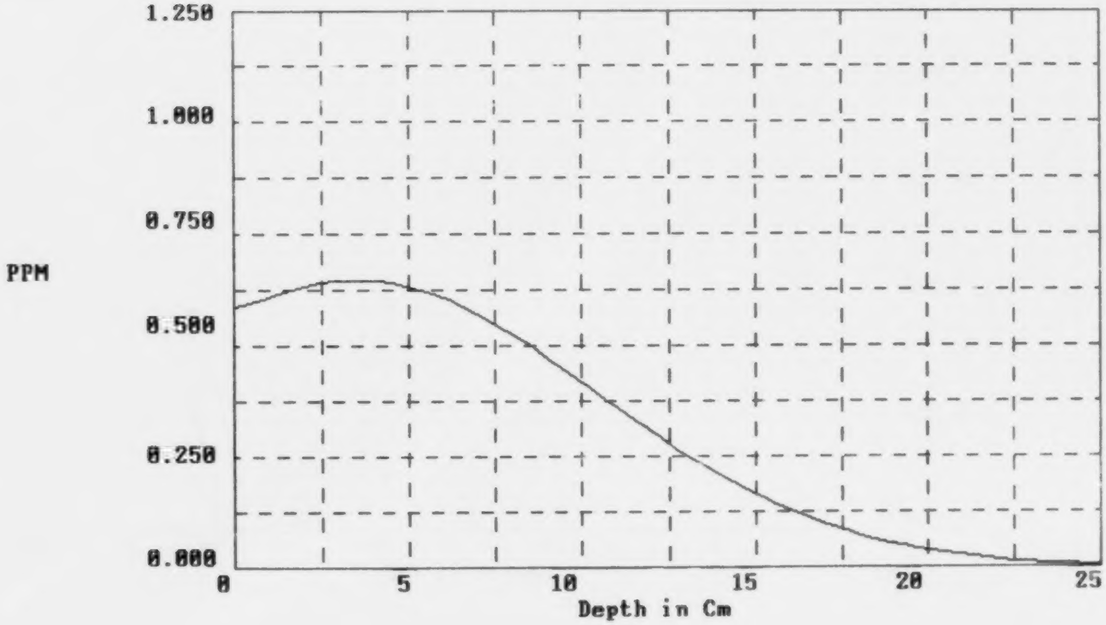

Figure 4-11--Leaching profiles of imazapyr in sandy loam soil after 6 cm of percolated water

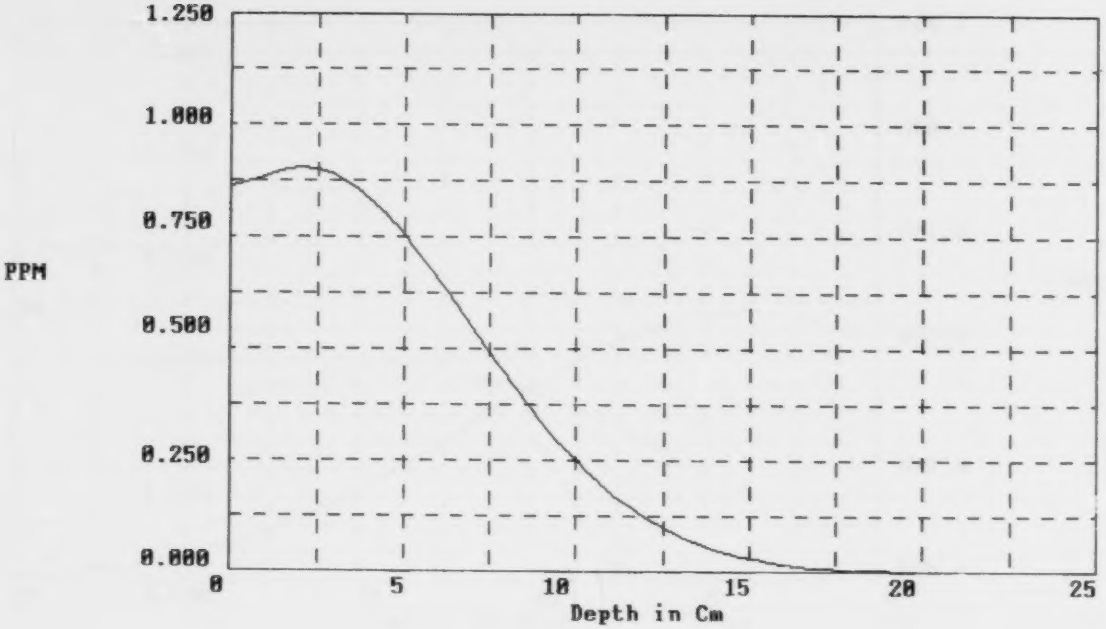

Figure 4-12--Leaching profiles of light fuel oil in sandy loam soil after 6 cm of percolated water

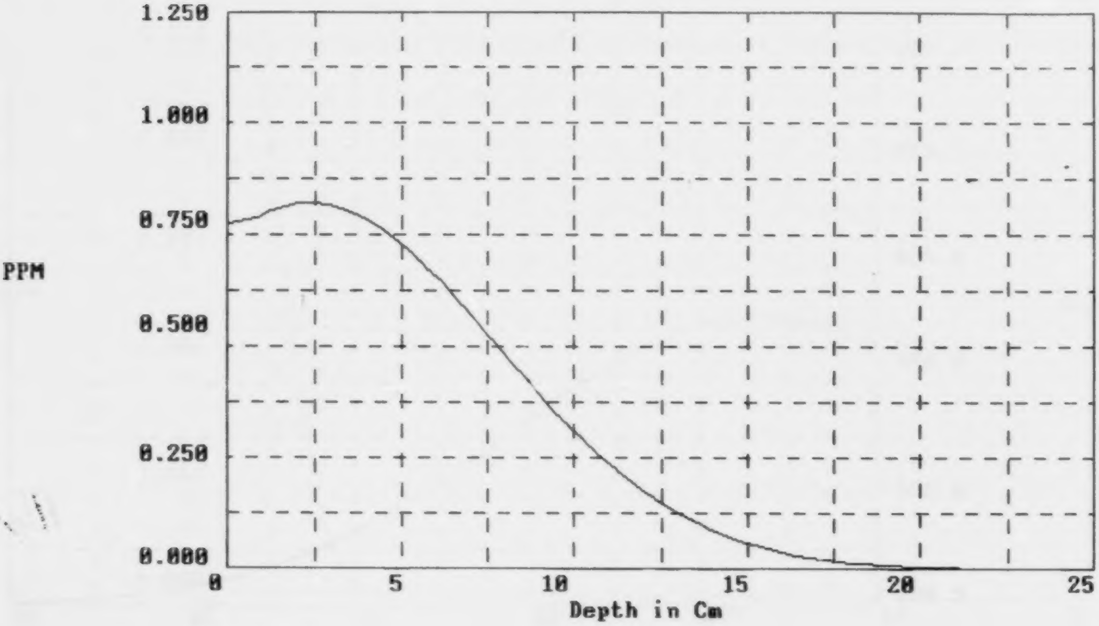

Figure 4-13--Leaching profiles of picloram in sandy loam soil after 6 cm of percolated water

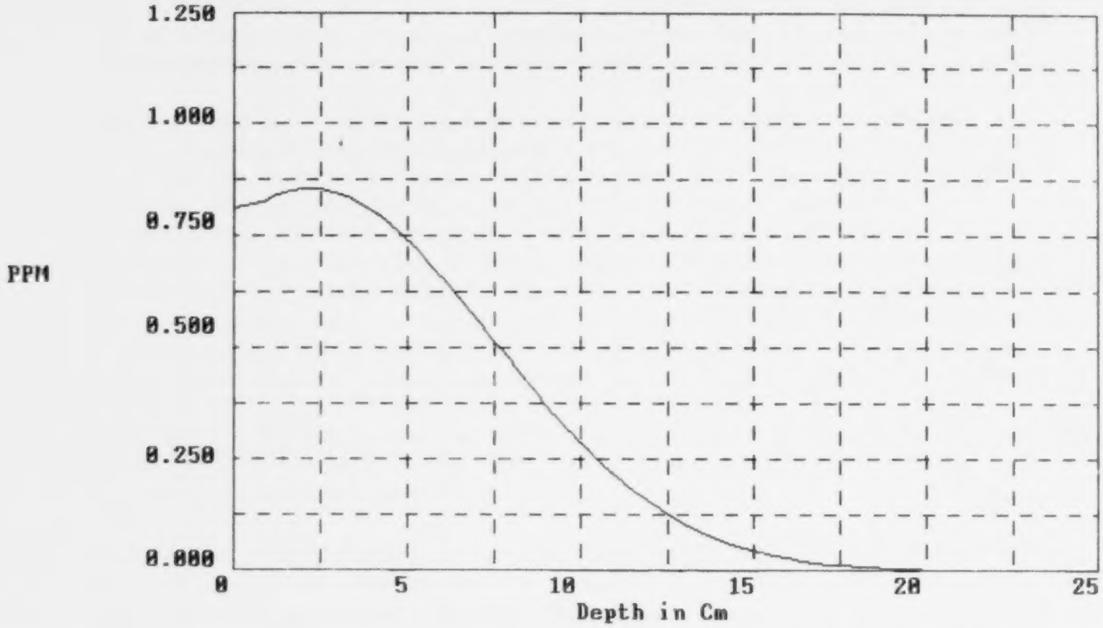

Figure 4-14--Leaching profiles of sulfometuron methyl in sandy loam soil after 6 cm of percolated water

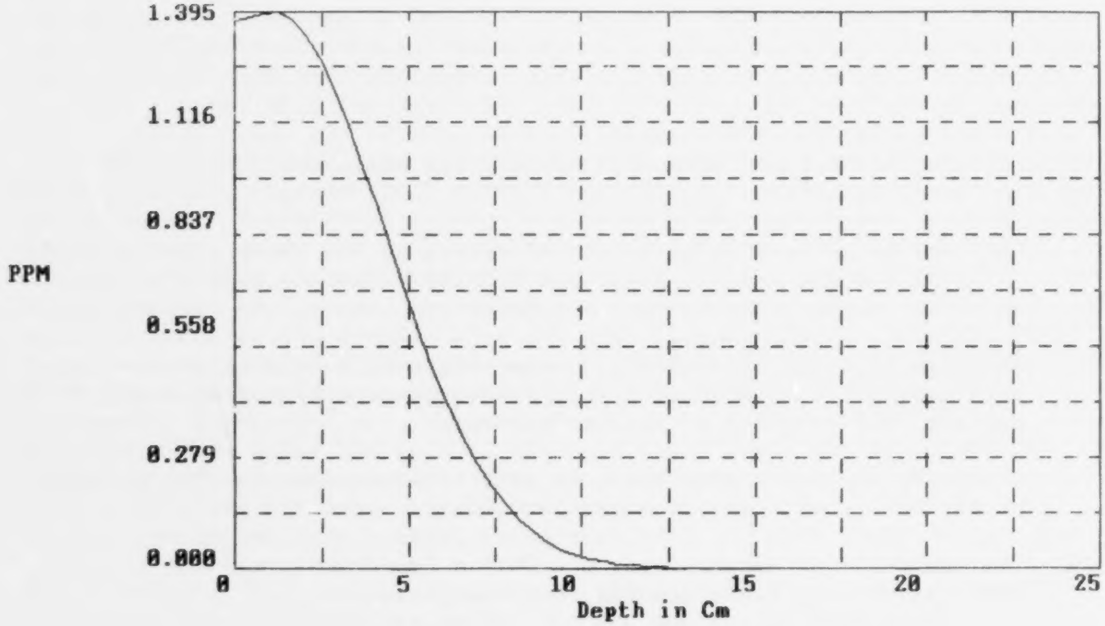

Figure 4-15--Leaching profiles of tebuthiuron in sandy loam soil after 6 cm of percolated water

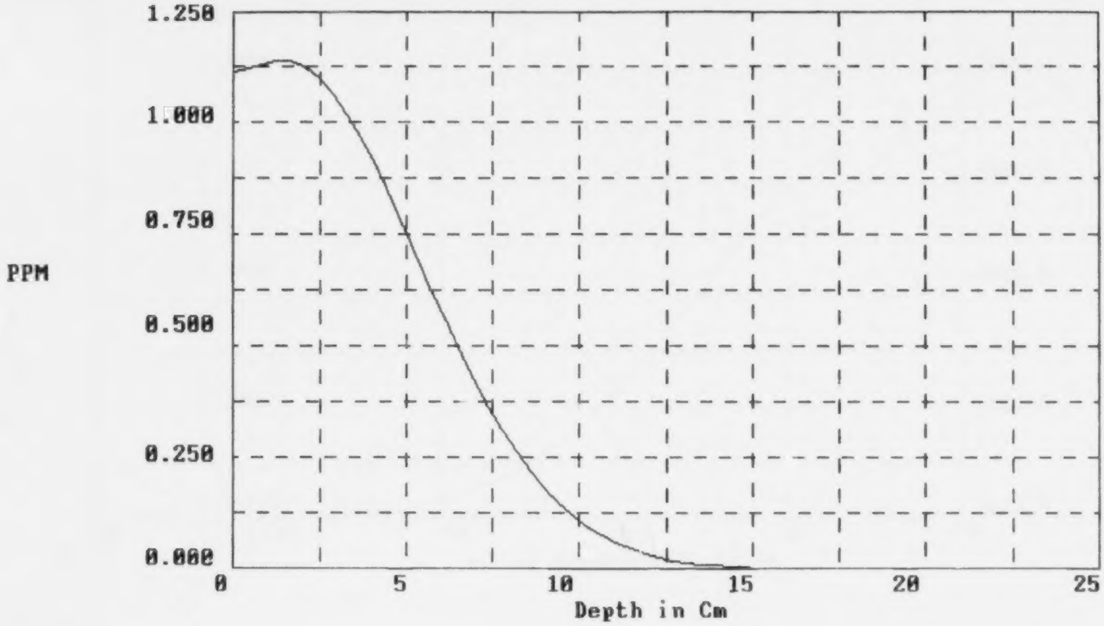

Figure 4-16--Leaching profiles of triclopyr in sandy loam soil after 6 cm of percolated water

aquifer is several hundreds of meters thick, and it serves as the primary source of water for both irrigation and drinking. The Floridan aquifer is much less likely to be contaminated with herbicides than the unconfined sand aquifer.

The following assumptions were used to simulate movement of herbicide through the sand aquifer:

(1) The aquifer is made up of saturated sands, 18.3 m thick. The porosity is 0.437 and the transmissivity is 0.00106 m^2 per second. The water table is assumed to be close (approximately 1 m) to the soil surface.

(2) The simulated area is 0.56 km^2 containing a 16.2-ha area treated with herbicide. The concentrations were calculated for square 1.01 ha cells (100.6 by 100.6 m) within this area.

(3) Ground-water flow within the area is at a steady state. The gradient of the water table is 5.4 m per 1000 m.

(4) The longitudinal dispersivity was 30.5 m (moderate), and the lateral dispersivity was 0.3 times the longitudinal.

(5) One pumping well (8.5 l/second) was located within the modeled area 200 m downgradient from the treatment site, but it had only a small effect on the predicted concentrations.

(6) The initial mass of herbicide in the aquifer under the treated sites was calculated using the maximum application rates anticipated to be used by the Forest Service, multiplied by the fraction leaching 10 percent of the time calculated previously using the LEACH methodology. As described in a previous section, these leaching predictions were developed for a typical southeastern coastal plain site with sandy loam soils. The site has moderately high rainfall and leaching potential. A level sand site is expected to have a somewhat greater leaching potential, but no purely sand soils were included among the standard sites considered in the LEACH Handbook.

(7) Time zero in the simulation occurs after leaching has occured, and the herbicide is assumed to be vertically mixed within the aquifer. This may require several months.

(8) Half-lives were assumed to be the same as those used for the LEACH methodology, but adsorption was assumed to be zero because the aquifer contains little organic matter and clay.

(9) Simulations were performed for all of the herbicides and also for benzene, representing the relatively soluble aromatic fraction of the kerosene contained in the ester formulations.

The results are shown in figures 4-17 and 4-18 for dicamba at the time of initial leaching and 0.2 years later. The initial concentration is 0.005 ppm, but the concentration in cells adjacent to the treated sites is less than 0.001 ppm at all times. Degradation is nearly complete at 0.2 year. The concentrations of hexazinone, imazapyr, and picloram at 0.2 years are shown in figures 4-19 through 4-21, respectively. The initial concentration under the treated site was about 0.004 ppm for hexazinone, and not more than 0.001 ppm for imazapyr and picloram. Most of these herbicides have degraded by 0.2 years and adjacent cells are never contaminated. Tebuthiuron is expected to be more persistent. Initial concentrations under the treated sites are 0.008 ppm, and figures 4-22 through 4-24 show the expected concentrations at 0.2, 1 and 2 years, respectively. At 2 years, the maximum concentration is only 0.002 ppm, and cells adjacent to the treated area are significantly less. Concentrations beyond two cells (201 m) downgradient of the treated sites do not exceed 0.001 ppm. Predicted concentrations of 2,4-D, 2,4-DP, fosamine, glyphosate, light fuel oil, sulfometuron methyl, and triclopyr are not measurable even under the treated site, so figures are not presented for these herbicides. Actual field data for sulfometuron methyl (Michael and Neary, 1987) and triclopyr (Neary, personal communication, 1987) in north Florida, under the same conditions as simulated, support these predictions.

EPA's Office of Groundwater Protection (1986a) recently surveyed State government agencies with responsibility for monitoring pesticide contamination of ground water. EPA's report summarizes the findings of 24 States that reported specific results. Detection of the herbicides used in

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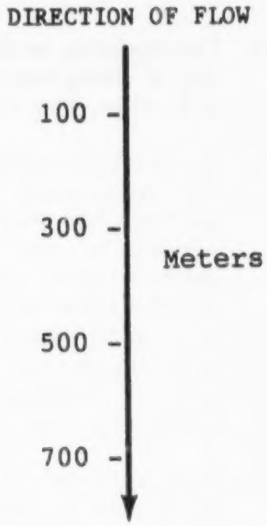

Note: Shading indicates treated area

[a] indicates location of a well

Figure 4-17--Two-dimensional ground water model predictions for dicamba at time zero in Florida near-surface aquifer, steady flow. Initial concentration (ave.) under treated area is 0.005 ppm.

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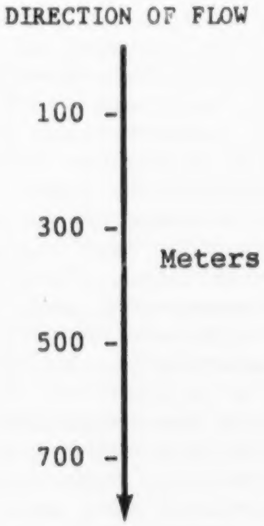

Note: Shading indicates treated area

[a] indicates location of a well

Figure 4-18--Two-dimensional ground water model predictions for dicamba at 0.2 years in Florida near-surface aquifer, steady flow. Initial concentration (ave.) under treated area is 0.005 ppm.

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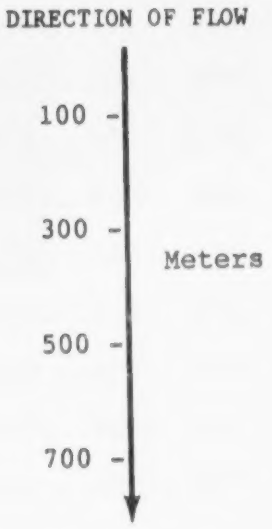

Note: Shading indicates treated area

[a] indicates location of a well

Figure 4-19--Two-dimensional ground water model predictions for hexazinone at 0.2 years in Florida near-surface aquifer, steady flow. Initial concentration (ave.) under treated area is 0.004 ppm.

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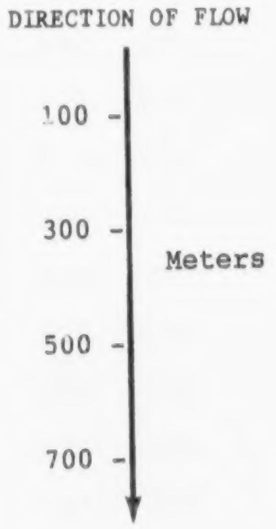
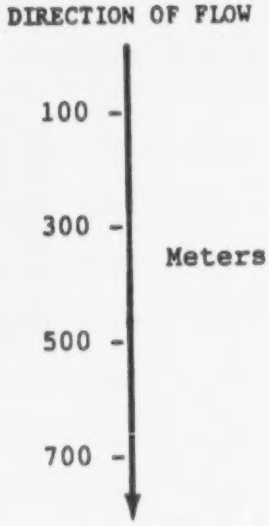


Note: Shading indicates treated area

[a] indicates location of a well

Figure 4-20--Two-dimensional ground water model predictions for imazapyr at 0.2 years in Florida near-surface aquifer, steady flow. Initial concentration (ave.) under treated area is 0.001 ppm.



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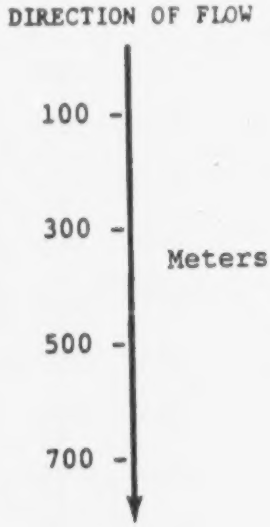
Note: Shading indicates treated area

[a] indicates location of a well

Figure 4-21--Two-dimensional ground water model predictions for picloram at 0.2 years in Florida near-surface aquifer, steady flow. Initial concentration (ave.) under treated area is less than 0.001 ppm.



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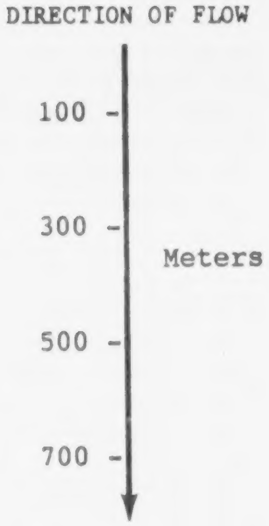
Note: Shading indicates treated area

[a] indicates location of a well

Figure 4-22--Two-dimensional ground water model predictions for tebuthiuron at 0.2 years in Florida near-surface aquifer, steady flow. Initial concentration (ave.) under treated area is 0.008 ppm.



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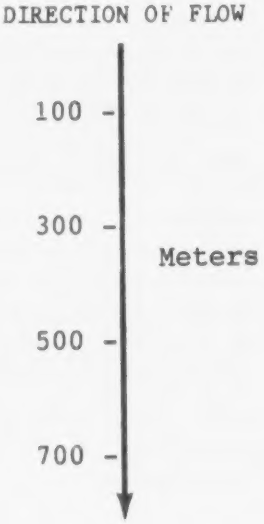
Note: Shading indicates treated area

[a] indicates location of a well

Figure 4-23--Two-dimensional ground water model predictions for tebuthiuron at 1 year in Florida near-surface aquifer, steady flow. Initial concentration (ave.) under treated area is 0.008 ppm.



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Note: Shading indicates treated area

[a] indicates location of a well

Figure 4-24--Two-dimensional ground water model predictions for tebuthiuron at 2 years in Florida near-surface aquifer, steady flow. Initial concentration (ave.) under treated area is 0.008 ppm.

Region 8 was very rare, but picloram was detected by four States and 2,4-D was reported by two. However, these surveys were generally conducted in areas of high agricultural use, where herbicide applications are more frequent and the amount used (lb a.i./ac) is greater per application. In the Southern Region, only about 10 percent of the herbicide-treated acres were treated with either of these chemicals.

In late 1984, EPA formed an agencywide Working Group on Pesticides in Ground Water. One objective of the working group was to complete an intensive review of existing information and scientific knowledge about the extent of pesticide contamination, its causes, and its potential impact on human health. The findings of the working group were summarized in "Pesticides in Ground Water: Background Document" (EPA, 1986b). The report points out that the potential sources of ground-water contamination include not only leaching of pesticides applied to the land surface, but also spills and leaks from storage areas or loading sites, disposal areas, and backflow to irrigation wells. The working group identified a list of pesticides that have been detected in ground water as a result of normal land application. None of the herbicides used by the Forest Service in Region 8 are on this list.

When the herbicides considered in this risk assessment have been detected in ground water, they generally are measured only in trace amounts that are not expected to be toxic. For example, EPA has established a tolerance of 0.1 ppm for 2,4-D in drinking water (Mullison, 1986), which is much higher than the trace amounts typically detected.

Based on both simulations and field monitoring studies, none of the herbicides considered in this analysis is considered a hazard to ground-water use. Subsequent consideration of exposures to humans from drinking water will be restricted to surface water bodies.

The Potential for Contamination of Surface Water by Runoff

Runoff of herbicide from the soil surface was estimated using a modification of the Haith (1980) model. The model was originally validated using pesticide runoff data derived from tests conducted in Georgia. The model considers adsorption and degradation to calculate a mass balance of pesticide in the top centimeter of soil. The pesticide in the surface soil is apportioned to adsorbed and dissolved phases, which are then available for loss as soil and water runoff from the treated plot. Runoff is calculated on a storm-by-storm basis. Runoff of both sediment (erosion) and water are calculated using standard Soil Conservation Service (SCS) techniques.

Erosion was calculated using the Universal Soil Loss Equation (Wischmeier and Smith, 1978). This equation was designed to predict average soil loss in runoff for specific soil, topographic, and vegetation conditions. The equation is based on a large amount of research data and has a long history of use. The basic equation is as follows:

$$A = RKLSCP$$

where:

A = the computed soil loss per unit area

R = the rainfall and runoff factor

K = the soil erodibility factor

L = the slope length factor

S = the slope steepness factor

C = the cover and management factor, relating soil loss under specific vegetation and management conditions to continuous fallow

P = the support practice factor, representing the effect of specific practices, for example contouring, that may reduce erosion relative to cultivation up- and down-slope

The Soil Conservation Service has given ample guidance on the selection of numerical values for the various factors in the equation (Wischmeier and Smith, 1978, and a variety of regional publications). Two additions to the equation have been made in the runoff model. First, rainfall erosivity has been calculated on a single-storm basis (Ateshian, 1974), as measured by 24-hour rainfalls. Second, provision has been made for the addition of a sediment delivery ratio factor to represent that fraction of sediment leaving a field that reaches a receiving water body. Any buffer area between treated plots and drainage channels, especially a well-vegetated buffer area, will substantially reduce the amount of eroded sediment reaching the channel. Sediment delivery ratios also generally decrease as the size of the drainage area increases (EPA, 1973). A Forest Service Handbook (1972) suggests a method for estimating the sediment delivery ratio as 1 minus the fraction for a minimum effective buffer strip:

$$SDR = 1 - \frac{\text{horizontal distance}}{\text{minimum effective buffer}}$$

where the horizontal distance is in feet measured from the edge of the stand to the nearest active channel, and the minimum effective buffer strip in feet is estimated to be 30 + 1.4 x (% slope to channel) (U.S. Forest Service, Region 8, 1987).

The volumes of runoff water were calculated by means of the Soil Conservation Service runoff curve number technique (USDA, 1972). Runoff curve numbers describe the tendency for rainwater to run off the land. The runoff curve number was used in the following equations to predict runoff volumes:

$$S = \frac{1000}{CN} - 10$$

and

$$Q = (P - 0.2S)^2/(P + 0.8S)$$

where:

CN = the runoff curve number

S = a retention parameter

P = the amount of rainfall (inches)

Q = the amount of runoff (inches)

The SCS National Engineering Handbook (USDA, 1972) provides guidance on the choice of runoff curve numbers. The handbook shows runoff curve numbers for various combinations of vegetation cover type and hydrologic soil group. All major soil series have been assigned by SCS to one of the four hydrologic soil groups, A through D. Soils in group A have an unusually low runoff potential, and soils in group D have an unusually high runoff potential.

The runoff and erosion prediction methods discussed above were combined with the following equations to predict herbicide runoff:

$$P_t = P_o \exp(-\alpha t)$$

$$P_t = A_t + D_t$$

$$A_t = [1/(1 + \Theta/(K_d \times \rho))] \times P_t$$

$$D_t = [1(1 + K_d \times \rho/\Theta)] \times P_t$$

$$PX_t = [X_t/100 \times \rho] \times A_t$$

$$PQ_t = [Q_t/R_t] \times D_t$$

where:

P_t = herbicide concentration at time t (g/ha)

P_o = initial herbicide concentration (g/ha)

α = herbicide degradation constant

t = elapsed time

A_t = adsorbed herbicide (g/ha)

Θ = available soil moisture capacity (cm/cm)

K_d = adsorption coefficient (mg/kg)/(mg/L)

ρ = soil bulk density (g/cm^3)

D_t = dissolved herbicide (g/ha)

PX_t = adsorbed herbicide lost in runoff (g/ha)

X_t = soil loss (tons/ha)

PQ_t = dissolved herbicide lost in runoff (g/ha)

Q_t = runoff (inches)

R_t = rainfall (inches)

The model was modified to account for adsorption to soil within a buffer zone. Calculations have also been added to the model to estimate the resulting concentrations in a pond fed by a small stream draining the treated land. The assumptions used for this scenario are intended to show the highest concentrations that could reasonably be expected to occur in a pond. The basic assumptions of the pond scenario are as follows:

(1) The pond is 0.2 ha in area and 1.5 m deep.

(2) The watershed is 40.5 ha in area.

(3) It is assumed that 50 percent of the watershed is treated with the herbicide.

(4) The treated acreage drains directly into the pond or stream without passing over a buffer zone, or after passing over buffer zones of 10 or 20 m. In practice, buffer zones are maintained around perennial water bodies (6.1 m minimum), but ephemeral streams may have no buffers.

(5) The volume of the pond does not change significantly; therefore, the outflow approximately equals the inflow.

(6) Processes within the pond, including degradation of the herbicide and sorption to bottom sediments, are not considered. Only the initial concentration is displayed here.

(7) Ground cover was assumed to be about 80 percent.

A required input to the model is an estimate of the fraction of the chemical in the top cm of soil. This was estimated assuming that 75 percent of the chemical reaches the soil surface, and then applying the one-dimensional leaching model (described previously under "Leaching Under Extreme Conditions"). The fraction was estimated after 5 cm (2 in) of water have percolated. More would be present on the surface at earlier times but most runoff is expected to occur after the surface soil is saturated. The fraction remaining in the top cm of soil was directly related to K_d. The surface fraction was estimated as 10 percent for chemicals with K_d less than 0.7, 15 percent for K_d between 0.7 and 1.5, and 57 percent for glyphosate, which has a K_d of approximately 16.5.

In order to investigate the variation in runoff concentrations under different conditions, 11 soil types were chosen to represent the dominant types encountered in the Region, and typical and maximum slopes were estimated for each. Table 4-10 lists the soil types, their surface texture, and organic matter contents. Runoff was simulated from each of these soil types for each herbicide at each buffer width. K_d for each herbicide-soil combination was determined using organic carbon partition coefficient (Koc) values based on Dean et al. (1984), or in some cases based on the K_d values presented earlier assuming that they refer to 1 percent organic carbon content. The simulations were performed using rainfalls that represent 2 and 5 year recurrence intervals, for typical and maximum slopes, and for 3 different buffer distances: 0, 10, and 20 m. Herbicides are not applied closer than 6.1 m to streams or ponds, but the case with no buffer represents ephemeral drainages that would flow during a large rainstorm.

The results are shown in tables 4-11 through 4-21. The runoff concentrations from the Cleveland and Pacolet soil are among the highest. These soils are in the steeper portions of the Piedmont and Appalachian Mountains and have the lowest organic matter content of the 11 soils compared in table 4-10. Although the largest runoff events carry the most herbicide, this is more than offset by dilution. It should be emphasized that the simulated situations are not expected to be typical, but they represent extreme events. The rainfall is assumed to occur the first day after application. In fact, it is standard Forest Service practice to avoid applications if rainfall is known to be imminent. Small localized showers are often not anticipated, but major storms can be predicted with reasonable accuracy.

Runoff predictions for glyphosate are not presented because it does not follow the model assumption that adsorption is controlled by organic matter content of soils. However, because of its strong adsorption and inactivation by soils (especially clays), glyphosate is not considered to be a potential problem in runoff.

Even though the estimated runoff concentrations are extreme, they are in general only a fraction of a part per million. In some cases, the runoff concentrations exceed those predicted for the maximum drift situation: 0.0006 to 0.022 ppm for a body of water 0.3 m (1 foot) deep. The runoff concentrations are not usually as great as those estimated for accidental spills into reservoirs (0.04 to 0.23 ppm), and they are much less than those estimated for spills into ponds (0.124 to 3.13 ppm). Consequently, margins of safety will be presented in section 5 only for people consuming water receiving drift and accidental spills.

The results of the runoff simulations are consistent with some field study results, but the simulation is likely to overestimate concentrations in many cases. Neary et al. (1983) measured hexazinone residues in runoff from four small watersheds in the Chattahoochee National Forest. During the first storm after application, residues in runoff from the treatment areas averaged 0.442 ppm, but the concentration of hexazinone (plus

Table 4-10

Representative soil types

Soil Type	Classification	Surface Texture	Percent Organic Matter
Coastal Plain			
Astatula	Hyperthermic, uncoated, typic quartzipsamment	Sand	1.0
Atmore	Coarse-loamy siliceous, thermic plinthic paleaquult	Silt loam	1.5
Benndale	Coarse-loamy siliceous, thermic typic paleudult	Fine sandy loam	3.0
Providence	Fine-silty, mixed, thermic typic fragiudalf	Silt loam loam	4.0
Smithdale	Fine-loamy siliceous, thermic typic paleudult	Sandy loam	3.0
Piedmont			
Pacolet	Clayey, kaolinitic, thermic typic hapludult	Sandy loam	1.0
Mountains			
Cleveland	Loamy, mixed, mesic lithic dystrochrept	Sandy loam	1.0
Edneytown	Fine-loamy, mixed, mesic typic hapludult	Fine sandy loam	2.0
Edneyville	Coarse-loamy, mixed mesic typic dystrochrept	Fine sandy loam	2.0
Sylco	Loamy-skeletal, mixed, mesic typic dystrochrept (rock content >35%)	Gravelly silt loam	3.0
Tusquitee	Coarse-loamy, mixed, mesic umbric dystrochrept	Loam	6.0

Table 4-11

Runoff model predictions for benzene

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm) Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.000	0.000	0.000
Atmore	5.5	2.4	2.0	0.000	0.000	0.000
Atmore	7.5	4.0	2.0	0.000	0.000	0.000
Benndale	4.8	0.9	5.0	0.000	0.000	0.000
Benndale	6.3	1.7	5.0	0.000	0.000	0.000
Benndale	4.8	0.9	10.0	0.000	0.000	0.000
Benndale	6.3	1.7	10.0	0.000	0.000	0.000
Providence	4.8	1.9	8.0	0.000	0.000	0.000
Providence	6.3	3.0	8.0	0.000	0.000	0.000
Providence	4.8	1.9	20.0	0.000	0.000	0.000
Providence	6.3	3.0	20.0	0.000	0.000	0.000
Smithdale	4.8	0.9	15.0	0.000	0.000	0.000
Smithdale	6.3	1.7	15.0	0.000	0.000	0.000
Smithdale	4.8	0.9	30.0	0.000	0.000	0.000
Smithdale	6.3	1.7	30.0	0.000	0.000	0.000
Pacolet	3.8	0.5	10.0	0.000	0.000	0.000
Pacolet	5.0	1.0	10.0	0.000	0.000	0.000
Pacolet	3.8	0.5	25.0	0.000	0.000	0.000
Pacolet	5.0	1.0	25.0	0.000	0.000	0.000
Cleveland	5.0	1.0	20.0	0.000	0.000	0.000
Cleveland	7.0	2.1	20.0	0.000	0.000	0.000
Cleveland	5.0	1.0	45.0	0.000	0.000	0.000
Cleveland	7.0	2.1	45.0	0.000	0.000	0.000
Edneytown	5.0	1.0	20.0	0.000	0.000	0.000
Edneytown	7.0	2.1	20.0	0.000	0.000	0.000
Edneytown	5.0	1.0	40.0	0.000	0.000	0.000
Edneytown	7.0	2.1	40.0	0.000	0.000	0.000
Edneyville	5.0	1.0	20.0	0.000	0.000	0.000
Edneyville	7.0	2.1	20.0	0.000	0.000	0.000
Edneyville	5.0	1.0	40.0	0.000	0.000	0.000
Edneyville	7.0	2.1	40.0	0.000	0.000	0.000
Sylco	4.0	0.5	25.0	0.000	0.000	0.000
Sylco	5.0	1.0	25.0	0.000	0.000	0.000
Sylco	4.0	0.5	45.0	0.000	0.000	0.000
Sylco	5.0	1.0	45.0	0.000	0.000	0.000
Tusquitee	5.0	1.0	15.0	0.000	0.000	0.000
Tusquitee	7.0	2.1	15.0	0.000	0.000	0.000
Tusquitee	5.0	1.0	35.0	0.000	0.000	0.000
Tusquitee	7.0	2.1	35.0	0.000	0.000	0.000

Table 4-12

Runoff model predictions for 2,4-D

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.003	0.001	0.001
Atmore	5.5	2.4	2.0	0.142	0.141	0.141
Atmore	7.5	4.0	2.0	0.104	0.104	0.104
Benndale	4.8	0.9	5.0	0.108	0.105	0.105
Benndale	6.3	1.7	5.0	0.087	0.084	0.084
Benndale	4.8	0.9	10.0	0.112	0.107	0.105
Benndale	6.3	1.7	10.0	0.091	0.086	0.084
Providence	4.8	1.9	8.0	0.113	0.108	0.107
Providence	6.3	3.0	8.0	0.088	0.083	0.081
Providence	4.8	1.9	20.0	0.133	0.118	0.107
Providence	6.3	3.0	20.0	0.111	0.094	0.081
Smithdale	4.8	0.9	15.0	0.120	0.110	0.105
Smithdale	6.3	1.7	15.0	0.099	0.089	0.084
Smithdale	4.8	0.9	30.0	0.152	0.130	0.109
Smithdale	6.3	1.7	30.0	0.131	0.109	0.088
Pacolet	3.8	0.5	10.0	0.172	0.169	0.168
Pacolet	5.0	1.0	10.0	0.162	0.159	0.158
Pacolet	3.8	0.5	25.0	0.185	0.177	0.168
Pacolet	5.0	1.0	25.0	0.176	0.167	0.158
Cleveland	5.0	1.0	20.0	0.170	0.163	0.158
Cleveland	7.0	2.1	20.0	0.130	0.122	0.117
Cleveland	5.0	1.0	45.0	0.204	0.188	0.171
Cleveland	7.0	2.1	45.0	0.164	0.147	0.131
Edneytown	5.0	1.0	20.0	0.142	0.132	0.124
Edneytown	7.0	2.1	20.0	0.110	0.100	0.092
Edneytown	5.0	1.0	40.0	0.180	0.159	0.137
Edneytown	7.0	2.1	40.0	0.148	0.127	0.105
Edneyville	5.0	1.0	20.0	0.142	0.132	0.124
Edneyville	7.0	2.1	20.0	0.110	0.100	0.092
Edneyville	5.0	1.0	40.0	0.180	0.159	0.137
Edneyville	7.0	2.1	40.0	0.148	0.127	0.105
Sylco	4.0	0.5	25.0	0.132	0.130	0.127
Sylco	5.0	1.0	25.0	0.123	0.121	0.118
Sylco	4.0	0.5	45.0	0.138	0.135	0.131
Sylco	5.0	1.0	45.0	0.130	0.126	0.122
Tusquitee	5.0	1.0	15.0	0.101	0.081	0.070
Tusquitee	7.0	2.1	15.0	0.083	0.063	0.052
Tusquitee	5.0	1.0	35.0	0.194	0.142	0.090
Tusquitee	7.0	2.1	35.0	0.177	0.125	0.073

Table 4-13

Runoff model predictions for 2-4-DP

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.003	0.001	0.001
Atmore	5.5	2.4	2.0	0.080	0.079	0.079
Atmore	7.5	4.0	2.0	0.059	0.058	0.058
Benndale	4.8	0.9	5.0	0.051	0.048	0.048
Benndale	6.3	1.7	5.0	0.042	0.039	0.038
Benndale	4.8	0.9	10.0	0.056	0.050	0.048
Benndale	6.3	1.7	10.0	0.047	0.041	0.038
Providence	4.8	1.9	8.0	0.055	0.049	0.048
Providence	6.3	3.0	8.0	0.045	0.038	0.037
Providence	4.8	1.9	20.0	0.078	0.061	0.048
Providence	6.3	3.0	20.0	0.070	0.051	0.037
Smithdale	4.8	0.9	15.0	0.065	0.054	0.048
Smithdale	6.3	1.7	15.0	0.056	0.045	0.038
Smithdale	4.8	0.9	30.0	0.102	0.077	0.052
Smithdale	6.3	1.7	30.0	0.092	0.068	0.043
Pacolet	3.8	0.5	10.0	0.104	0.099	0.098
Pacolet	5.0	1.0	10.0	0.098	0.093	0.092
Pacolet	3.8	0.5	25.0	0.123	0.110	0.098
Pacolet	5.0	1.0	25.0	0.118	0.105	0.092
Cleveland	5.0	1.0	20.0	0.110	0.100	0.092
Cleveland	7.0	2.1	20.0	0.086	0.076	0.068
Cleveland	5.0	1.0	45.0	0.159	0.135	0.111
Cleveland	7.0	2.1	45.0	0.136	0.112	0.088
Edneytown	5.0	1.0	20.0	0.084	0.072	0.062
Edneytown	7.0	2.1	20.0	0.069	0.056	0.046
Edneytown	5.0	1.0	40.0	0.132	0.105	0.078
Edneytown	7.0	2.1	40.0	0.116	0.089	0.062
Edneyville	5.0	1.0	20.0	0.084	0.072	0.062
Edneyville	7.0	2.1	20.0	0.069	0.056	0.046
Edneyville	5.0	1.0	40.0	0.132	0.105	0.078
Edneyville	7.0	2.1	40.0	0.116	0.089	0.062
Sylco	4.0	0.5	25.0	0.067	0.065	0.062
Sylco	5.0	1.0	25.0	0.063	0.060	0.058
Sylco	4.0	0.5	45.0	0.075	0.071	0.066
Sylco	5.0	1.0	45.0	0.071	0.066	0.062
Tusquitee	5.0	1.0	15.0	0.060	0.040	0.028
Tusquitee	7.0	2.1	15.0	0.053	0.032	0.021
Tusquitee	5.0	1.0	35.0	0.155	0.102	0.049
Tusquitee	7.0	2.1	35.0	0.148	0.095	0.042

Table 4-14

Runoff model predictions for dicamba

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.001	0.001	0.001
Atmore	5.5	2.4	2.0	0.083	0.083	0.083
Atmore	7.5	4.0	2.0	0.061	0.061	0.061
Benndale	4.8	0.9	5.0	0.085	0.084	0.084
Benndale	6.3	1.7	5.0	0.068	0.068	0.068
Benndale	4.8	0.9	10.0	0.085	0.085	0.084
Benndale	6.3	1.7	10.0	0.068	0.068	0.068
Providence	4.8	1.9	8.0	0.089	0.088	0.088
Providence	6.3	3.0	8.0	0.068	0.068	0.067
Providence	4.8	1.9	20.0	0.091	0.089	0.088
Providence	6.3	3.0	20.0	0.070	0.069	0.067
Smithdale	4.8	0.9	15.0	0.086	0.085	0.084
Smithdale	6.3	1.7	15.0	0.069	0.068	0.068
Smithdale	4.8	0.9	30.0	0.089	0.087	0.085
Smithdale	6.3	1.7	30.0	0.072	0.070	0.068
Pacolet	3.8	0.5	10.0	0.095	0.095	0.095
Pacolet	5.0	1.0	10.0	0.089	0.089	0.089
Pacolet	3.8	0.5	25.0	0.096	0.095	0.095
Pacolet	5.0	1.0	25.0	0.090	0.089	0.089
Cleveland	5.0	1.0	20.0	0.090	0.089	0.089
Cleveland	7.0	2.1	20.0	0.067	0.066	0.066
Cleveland	5.0	1.0	45.0	0.092	0.091	0.090
Cleveland	7.0	2.1	45.0	0.069	0.068	0.067
Edneytown	5.0	1.0	20.0	0.087	0.086	0.085
Edneytown	7.0	2.1	20.0	0.065	0.064	0.063
Edneytown	5.0	1.0	40.0	0.090	0.088	0.086
Edneytown	7.0	2.1	40.0	0.068	0.066	0.064
Edneyville	5.0	1.0	20.0	0.087	0.086	0.085
Edneyville	7.0	2.1	20.0	0.065	0.064	0.063
Edneyville	5.0	1.0	40.0	0.090	0.088	0.086
Edneyville	7.0	2.1	40.0	0.068	0.066	0.064
Sylco	4.0	0.5	25.0	0.092	0.091	0.091
Sylco	5.0	1.0	25.0	0.085	0.085	0.085
Sylco	4.0	0.5	45.0	0.092	0.092	0.092
Sylco	5.0	1.0	45.0	0.086	0.085	0.085
Tusquitee	5.0	1.0	15.0	0.079	0.076	0.075
Tusquitee	7.0	2.1	15.0	0.059	0.057	0.056
Tusquitee	5.0	1.0	35.0	0.090	0.084	0.077
Tusquitee	7.0	2.1	35.0	0.071	0.064	0.058

Table 4-15

Runoff model predictions for fosamine

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.045	0.002	0.000
Atmore	5.5	2.4	2.0	0.057	0.041	0.040
Atmore	7.5	4.0	2.0	0.049	0.030	0.030
Benndale	4.8	0.9	5.0	0.069	0.023	0.017
Benndale	6.3	1.7	5.0	0.065	0.020	0.014
Benndale	4.8	0.9	10.0	0.149	0.050	0.017
Benndale	6.3	1.7	10.0	0.145	0.047	0.014
Providence	4.8	1.9	8.0	0.124	0.039	0.017
Providence	6.3	3.0	8.0	0.134	0.037	0.013
Providence	4.8	1.9	20.0	0.461	0.209	0.017
Providence	6.3	3.0	20.0	0.515	0.230	0.013
Smithdale	4.8	0.9	15.0	0.285	0.112	0.017
Smithdale	6.3	1.7	15.0	0.281	0.108	0.014
Smithdale	4.8	0.9	30.0	0.845	0.465	0.086
Smithdale	6.3	1.7	30.0	0.839	0.461	0.083
Pacolet	3.8	0.5	10.0	0.189	0.087	0.053
Pacolet	5.0	1.0	10.0	0.191	0.086	0.051
Pacolet	3.8	0.5	25.0	0.631	0.337	0.053
Pacolet	5.0	1.0	25.0	0.652	0.347	0.051
Cleveland	5.0	1.0	20.0	0.469	0.231	0.051
Cleveland	7.0	2.1	20.0	0.457	0.219	0.038
Cleveland	5.0	1.0	45.0	1.602	1.052	0.501
Cleveland	7.0	2.1	45.0	1.597	1.043	0.490
Edneytown	5.0	1.0	20.0	0.415	0.193	0.025
Edneytown	7.0	2.1	20.0	0.410	0.188	0.019
Edneytown	5.0	1.0	40.0	1.233	0.769	0.306
Edneytown	7.0	2.1	40.0	1.232	0.766	0.301
Edneyville	5.0	1.0	20.0	0.415	0.193	0.025
Edneyville	7.0	2.1	20.0	0.410	0.188	0.019
Edneyville	5.0	1.0	40.0	1.233	0.769	0.306
Edneyville	7.0	2.1	40.0	1.232	0.766	0.301
Sylco	4.0	0.5	25.0	0.111	0.066	0.023
Sylco	5.0	1.0	25.0	0.111	0.066	0.022
Sylco	4.0	0.5	45.0	0.246	0.167	0.087
Sylco	5.0	1.0	45.0	0.250	0.169	0.088
Tusquitee	5.0	1.0	15.0	0.440	0.161	0.008
Tusquitee	7.0	2.1	15.0	0.440	0.159	0.007
Tusquitee	5.0	1.0	35.0	1.718	1.004	0.290
Tusquitee	7.0	2.1	35.0	1.724	1.006	0.289

Table 4-16

Runoff model predictions for hexazinone

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.002	0.001	0.001
Atmore	5.5	2.4	2.0	0.121	0.121	0.121
Atmore	7.5	4.0	2.0	0.089	0.088	0.088
Benndale	4.8	0.9	5.0	0.092	0.089	0.089
Benndale	6.3	1.7	5.0	0.074	0.072	0.071
Benndale	4.8	0.9	10.0	0.096	0.091	0.089
Benndale	6.3	1.7	10.0	0.078	0.073	0.071
Providence	4.8	1.9	8.0	0.096	0.092	0.091
Providence	6.3	3.0	8.0	0.075	0.070	0.069
Providence	4.8	1.9	20.0	0.113	0.100	0.091
Providence	6.3	3.0	20.0	0.095	0.080	0.069
Smithdale	4.8	0.9	15.0	0.102	0.094	0.089
Smithdale	6.3	1.7	15.0	0.085	0.076	0.071
Smithdale	4.8	0.9	30.0	0.130	0.111	0.092
Smithdale	6.3	1.7	30.0	0.112	0.093	0.075
Pacolet	3.8	0.5	10.0	0.147	0.144	0.143
Pacolet	5.0	1.0	10.0	0.138	0.136	0.135
Pacolet	3.8	0.5	25.0	0.159	0.151	0.143
Pacolet	5.0	1.0	25.0	0.150	0.142	0.135
Cleveland	5.0	1.0	20.0	0.146	0.139	0.135
Cleveland	7.0	2.1	20.0	0.111	0.105	0.100
Cleveland	5.0	1.0	45.0	0.175	0.161	0.146
Cleveland	7.0	2.1	45.0	0.141	0.126	0.112
Edneytown	5.0	1.0	20.0	0.121	0.112	0.106
Edneytown	7.0	2.1	20.0	0.094	0.085	0.078
Edneytown	5.0	1.0	40.0	0.154	0.136	0.117
Edneytown	7.0	2.1	40.0	0.127	0.108	0.090
Edneyville	5.0	1.0	20.0	0.121	0.112	0.106
Edneyville	7.0	2.1	20.0	0.094	0.085	0.078
Edneyville	5.0	1.0	40.0	0.154	0.136	0.117
Edneyville	7.0	2.1	40.0	0.127	0.108	0.090
Sylco	4.0	0.5	25.0	0.112	0.110	0.108
Sylco	5.0	1.0	25.0	0.104	0.103	0.101
Sylco	4.0	0.5	45.0	0.118	0.115	0.111
Sylco	5.0	1.0	45.0	0.110	0.107	0.104
Tusquitee	5.0	1.0	15.0	0.086	0.069	0.059
Tusquitee	7.0	2.1	15.0	0.071	0.054	0.044
Tusquitee	5.0	1.0	35.0	0.167	0.122	0.077
Tusquitee	7.0	2.1	35.0	0.152	0.107	0.062

Table 4-17

Runoff model predictions for imazapyr

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.001	0.000	0.000
Atmore	5.5	2.4	2.0	0.027	0.027	0.027
Atmore	7.5	4.0	2.0	0.020	0.020	0.020
Benndale	4.8	0.9	5.0	0.019	0.019	0.018
Benndale	6.3	1.7	5.0	0.016	0.015	0.015
Benndale	4.8	0.9	10.0	0.020	0.019	0.018
Benndale	6.3	1.7	10.0	0.017	0.015	0.015
Providence	4.8	1.9	8.0	0.020	0.019	0.019
Providence	6.3	3.0	8.0	0.016	0.015	0.014
Providence	4.8	1.9	20.0	0.025	0.021	0.019
Providence	6.3	3.0	20.0	0.022	0.017	0.014
Smithdale	4.8	0.9	15.0	0.022	0.020	0.018
Smithdale	6.3	1.7	15.0	0.019	0.016	0.015
Smithdale	4.8	0.9	30.0	0.030	0.025	0.019
Smithdale	6.3	1.7	30.0	0.026	0.021	0.016
Pacolet	3.8	0.5	10.0	0.034	0.033	0.032
Pacolet	5.0	1.0	10.0	0.032	0.031	0.030
Pacolet	3.8	0.5	25.0	0.037	0.035	0.032
Pacolet	5.0	1.0	25.0	0.035	0.033	0.030
Cleveland	5.0	1.0	20.0	0.034	0.032	0.030
Cleveland	7.0	2.1	20.0	0.026	0.024	0.023
Cleveland	5.0	1.0	45.0	0.043	0.039	0.034
Cleveland	7.0	2.1	45.0	0.035	0.031	0.026
Edneytown	5.0	1.0	20.0	0.027	0.025	0.023
Edneytown	7.0	2.1	20.0	0.021	0.019	0.017
Edneytown	5.0	1.0	40.0	0.037	0.031	0.026
Edneytown	7.0	2.1	40.0	0.031	0.026	0.020
Edneyville	5.0	1.0	20.0	0.027	0.025	0.023
Edneyville	7.0	2.1	20.0	0.021	0.019	0.017
Edneyville	5.0	1.0	40.0	0.037	0.031	0.026
Edneyville	7.0	2.1	40.0	0.031	0.026	0.020
Sylco	4.0	0.5	25.0	0.024	0.024	0.023
Sylco	5.0	1.0	25.0	0.023	0.022	0.021
Sylco	4.0	0.5	45.0	0.026	0.025	0.024
Sylco	5.0	1.0	45.0	0.024	0.023	0.022
Tusquitee	5.0	1.0	15.0	0.019	0.014	0.012
Tusquitee	7.0	2.1	15.0	0.016	0.011	0.009
Tusquitee	5.0	1.0	35.0	0.041	0.029	0.017
Tusquitee	7.0	2.1	35.0	0.038	0.026	0.014

Table 4-18

Runoff model predictions for picloram

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.001	0.000	0.000
Atmore	5.5	2.4	2.0	0.026	0.026	0.026
Atmore	7.5	4.0	2.0	0.019	0.019	0.019
Benndale	4.8	0.9	5.0	0.019	0.018	0.018
Benndale	6.3	1.7	5.0	0.015	0.015	0.015
Benndale	4.8	0.9	10.0	0.020	0.019	0.018
Benndale	6.3	1.7	10.0	0.016	0.015	0.015
Providence	4.8	1.9	8.0	0.020	0.019	0.019
Providence	6.3	3.0	8.0	0.016	0.014	0.014
Providence	4.8	1.9	20.0	0.024	0.021	0.019
Providence	6.3	3.0	20.0	0.021	0.017	0.014
Smithdale	4.8	0.9	15.0	0.022	0.020	0.018
Smithdale	6.3	1.7	15.0	0.018	0.016	0.015
Smithdale	4.8	0.9	30.0	0.029	0.024	0.019
Smithdale	6.3	1.7	30.0	0.025	0.020	0.016
Pacolet	3.8	0.5	10.0	0.032	0.032	0.031
Pacolet	5.0	1.0	10.0	0.031	0.030	0.030
Pacolet	3.8	0.5	25.0	0.036	0.034	0.031
Pacolet	5.0	1.0	25.0	0.034	0.032	0.030
Cleveland	5.0	1.0	20.0	0.033	0.031	0.030
Cleveland	7.0	2.1	20.0	0.025	0.023	0.022
Cleveland	5.0	1.0	45.0	0.041	0.037	0.033
Cleveland	7.0	2.1	45.0	0.033	0.029	0.025
Edneytown	5.0	1.0	20.0	0.027	0.024	0.022
Edneytown	7.0	2.1	20.0	0.021	0.018	0.017
Edneytown	5.0	1.0	40.0	0.035	0.030	0.025
Edneytown	7.0	2.1	40.0	0.030	0.025	0.020
Edneyville	5.0	1.0	20.0	0.027	0.024	0.022
Edneyville	7.0	2.1	20.0	0.021	0.018	0.017
Edneyville	5.0	1.0	40.0	0.035	0.030	0.025
Edneyville	7.0	2.1	40.0	0.030	0.025	0.020
Sylco	4.0	0.5	25.0	0.024	0.023	0.023
Sylco	5.0	1.0	25.0	0.022	0.022	0.021
Sylco	4.0	0.5	45.0	0.025	0.024	0.023
Sylco	5.0	1.0	45.0	0.024	0.023	0.022
Tusquitee	5.0	1.0	15.0	0.019	0.014	0.012
Tusquitee	7.0	2.1	15.0	0.016	0.011	0.009
Tusquitee	5.0	1.0	35.0	0.039	0.028	0.016
Tusquitee	7.0	2.1	35.0	0.036	0.025	0.013

Table 4-19

Runoff model predictions for sulfometuron methyl

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm) Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.000	0.000	0.000
Atmore	5.5	2.4	2.0	0.006	0.006	0.006
Atmore	7.5	4.0	2.0	0.004	0.004	0.004
Benndale	4.8	0.9	5.0	0.004	0.003	0.003
Benndale	6.3	1.7	5.0	0.003	0.003	0.003
Benndale	4.8	0.9	10.0	0.004	0.004	0.003
Benndale	6.3	1.7	10.0	0.004	0.003	0.003
Providence	4.8	1.9	8.0	0.004	0.003	0.003
Providence	6.3	3.0	8.0	0.003	0.003	0.003
Providence	4.8	1.9	20.0	0.006	0.005	0.003
Providence	6.3	3.0	20.0	0.006	0.004	0.003
Smithdale	4.8	0.9	15.0	0.005	0.004	0.003
Smithdale	6.3	1.7	15.0	0.004	0.003	0.003
Smithdale	4.8	0.9	30.0	0.009	0.006	0.004
Smithdale	6.3	1.7	30.0	0.008	0.006	0.003
Pacolet	3.8	0.5	10.0	0.008	0.008	0.007
Pacolet	5.0	1.0	10.0	0.008	0.007	0.007
Pacolet	3.8	0.5	25.0	0.010	0.009	0.007
Pacolet	5.0	1.0	25.0	0.010	0.008	0.007
Cleveland	5.0	1.0	20.0	0.009	0.008	0.007
Cleveland	7.0	2.1	20.0	0.007	0.006	0.005
Cleveland	5.0	1.0	45.0	0.014	0.012	0.009
Cleveland	7.0	2.1	45.0	0.013	0.010	0.007
Edneytown	5.0	1.0	20.0	0.007	0.005	0.004
Edneytown	7.0	2.1	20.0	0.006	0.004	0.003
Edneytown	5.0	1.0	40.0	0.012	0.009	0.006
Edneytown	7.0	2.1	40.0	0.011	0.008	0.005
Edneyville	5.0	1.0	20.0	0.007	0.005	0.004
Edneyville	7.0	2.1	20.0	0.006	0.004	0.003
Edneyville	5.0	1.0	40.0	0.012	0.009	0.006
Edneyville	7.0	2.1	40.0	0.011	0.008	0.005
Sylco	4.0	0.5	25.0	0.005	0.005	0.004
Sylco	5.0	1.0	25.0	0.005	0.004	0.004
Sylco	4.0	0.5	45.0	0.006	0.005	0.005
Sylco	5.0	1.0	45.0	0.005	0.005	0.004
Tusquitee	5.0	1.0	15.0	0.005	0.003	0.002
Tusquitee	7.0	2.1	15.0	0.005	0.003	0.001
Tusquitee	5.0	1.0	35.0	0.014	0.009	0.004
Tusquitee	7.0	2.1	35.0	0.014	0.009	0.003

Table 4-20

Runoff model predictions for tebuthiuron

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.007	0.001	0.000
Atmore	5.5	2.4	2.0	0.055	0.052	0.052
Atmore	7.5	4.0	2.0	0.041	0.039	0.038
Benndale	4.8	0.9	5.0	0.034	0.026	0.025
Benndale	6.3	1.7	5.0	0.029	0.021	0.020
Benndale	4.8	0.9	10.0	0.047	0.031	0.025
Benndale	6.3	1.7	10.0	0.042	0.026	0.020
Providence	4.8	1.9	8.0	0.043	0.028	0.025
Providence	6.3	3.0	8.0	0.039	0.023	0.019
Providence	4.8	1.9	20.0	0.100	0.057	0.025
Providence	6.3	3.0	20.0	0.103	0.055	0.019
Smithdale	4.8	0.9	15.0	0.070	0.041	0.025
Smithdale	6.3	1.7	15.0	0.065	0.036	0.020
Smithdale	4.8	0.9	30.0	0.164	0.100	0.036
Smithdale	6.3	1.7	30.0	0.158	0.095	0.032
Pacolet	3.8	0.5	10.0	0.089	0.074	0.069
Pacolet	5.0	1.0	10.0	0.086	0.070	0.065
Pacolet	3.8	0.5	25.0	0.155	0.111	0.069
Pacolet	5.0	1.0	25.0	0.155	0.109	0.065
Cleveland	5.0	1.0	20.0	0.127	0.092	0.065
Cleveland	7.0	2.1	20.0	0.111	0.075	0.048
Cleveland	5.0	1.0	45.0	0.298	0.215	0.132
Cleveland	7.0	2.1	45.0	0.282	0.199	0.116
Edneytown	5.0	1.0	20.0	0.099	0.063	0.035
Edneytown	7.0	2.1	20.0	0.090	0.054	0.026
Edneytown	5.0	1.0	40.0	0.232	0.157	0.081
Edneytown	7.0	2.1	40.0	0.224	0.148	0.072
Edneyville	5.0	1.0	20.0	0.099	0.063	0.035
Edneyville	7.0	2.1	20.0	0.090	0.054	0.026
Edneyville	5.0	1.0	40.0	0.232	0.157	0.081
Edneyville	7.0	2.1	40.0	0.224	0.148	0.072
Sylco	4.0	0.5	25.0	0.049	0.042	0.034
Sylco	5.0	1.0	25.0	0.047	0.039	0.032
Sylco	4.0	0.5	45.0	0.071	0.058	0.045
Sylco	5.0	1.0	45.0	0.069	0.056	0.043
Tusquitee	5.0	1.0	15.0	0.088	0.040	0.013
Tusquitee	7.0	2.1	15.0	0.085	0.036	0.010
Tusquitee	5.0	1.0	35.0	0.308	0.185	0.062
Tusquitee	7.0	2.1	35.0	0.306	0.182	0.059

Table 4-21

Runoff model predictions for triclopyr

	Rain (INS.)	Runoff (INS.)	Slope (%)	Concentration In Pond (ppm)		
				Buffer: 0 m	10 m	20 m
Astatula	4.8	0.0	3.0	0.000	0.000	0.000
Astatula	6.3	0.0	3.0	0.009	0.001	0.001
Atmore	5.5	2.4	2.0	0.099	0.096	0.096
Atmore	7.5	4.0	2.0	0.074	0.070	0.070
Benndale	4.8	0.9	5.0	0.058	0.049	0.048
Benndale	6.3	1.7	5.0	0.049	0.040	0.039
Benndale	4.8	0.9	10.0	0.074	0.055	0.048
Benndale	6.3	1.7	10.0	0.065	0.045	0.039
Providence	4.8	1.9	8.0	0.069	0.052	0.048
Providence	6.3	3.0	8.0	0.061	0.041	0.036
Providence	4.8	1.9	20.0	0.138	0.086	0.048
Providence	6.3	3.0	20.0	0.138	0.080	0.036
Smithdale	4.8	0.9	15.0	0.102	0.067	0.048
Smithdale	6.3	1.7	15.0	0.092	0.057	0.039
Smithdale	4.8	0.9	30.0	0.214	0.138	0.062
Smithdale	6.3	1.7	30.0	0.204	0.128	0.052
Pacolet	3.8	0.5	10.0	0.147	0.130	0.124
Pacolet	5.0	1.0	10.0	0.140	0.122	0.116
Pacolet	3.8	0.5	25.0	0.222	0.172	0.124
Pacolet	5.0	1.0	25.0	0.218	0.166	0.116
Cleveland	5.0	1.0	20.0	0.187	0.147	0.116
Cleveland	7.0	2.1	20.0	0.157	0.117	0.086
Cleveland	5.0	1.0	45.0	0.379	0.286	0.193
Cleveland	7.0	2.1	45.0	0.350	0.256	0.163
Edneytown	5.0	1.0	20.0	0.142	0.099	0.067
Edneytown	7.0	2.1	20.0	0.125	0.082	0.050
Edneytown	5.0	1.0	40.0	0.298	0.209	0.121
Edneytown	7.0	2.1	40.0	0.282	0.193	0.104
Edneyville	5.0	1.0	20.0	0.142	0.099	0.067
Edneyville	7.0	2.1	20.0	0.125	0.082	0.050
Edneyville	5.0	1.0	40.0	0.298	0.209	0.121
Edneyville	7.0	2.1	40.0	0.282	0.193	0.104
Sylco	4.0	0.5	25.0	0.083	0.074	0.065
Sylco	5.0	1.0	25.0	0.078	0.069	0.061
Sylco	4.0	0.5	45.0	0.109	0.093	0.078
Sylco	5.0	1.0	45.0	0.105	0.089	0.074
Tusquitee	5.0	1.0	15.0	0.117	0.058	0.026
Tusquitee	7.0	2.1	15.0	0.111	0.052	0.020
Tusquitee	5.0	1.0	35.0	0.386	0.235	0.085
Tusquitee	7.0	2.1	35.0	0.380	0.230	0.079

metabolites) in a second-order perennial stream below the treated watersheds did not exceed 0.044 ppm. Mayeux et al. (1984) measured maximum picloram concentrations of 0.048 and 0.25 ppm in runoff from a treated area during 2 successive years. Several other picloram runoff studies reviewed by Mullison (undated) have shown that concentrations can reach several ppm in runoff water if rainfall occurs soon after application. However, picloram concentrations decline rapidly with distance from the source, and with time after treatment. Concentrations in runoff from forested watersheds are often low (less than 0.01 ppm) or undetectable (for example, Neary et al., 1985). Measurements of sulfometuron methyl runoff from forest watersheds in Florida and Mississippi also showed measurable concentrations only close to the treatment sites when rainfall occured soon after application (Michael and Neary, 1987). At the Florida site with a small streamside management zone (<5 m), concentrations were less than 0.007 ppm. At the Mississippi site with a larger streamside management zone, concentrations peaked at 0.044 ppm.

Herbicide Exposures From Brown-and-Burn Operations

In Region 8, approximately 3,850 ha (9,500 ac) per year are typically treated with herbicides, followed by a prescribed burn (brown-and-burn); the maximum area treated and subsequently burned in a year is less than 6,720 ha (16,600 ac). Exposures to herbicide residues released from burning treated vegetation were estimated for workers onsite. Usually, in brown-and-burn operations, the vegetation at a selected site is burned 45 to 180 days after treatment with a selected herbicide. The time between application and burning varies with the herbicide used and the method of application. The minimum time between herbicide application and a prescribed burn is 30 days.

In order to model exposure to herbicide residues from brown-and-burn operations, the following assumptions were used:

(1) There are 44,500 kg (98,000 lb) of fuel per ha (2.5 ac), and 40 percent (17,800 kg) of it is burned (Anderson, 1982).

(2) Smoke density is 30 mg/m^3 at 100 m (328 ft) (USDA Forest Service, 1976).

(3) 50 g (0.11 lb) of smoke are produced for each kg (2.2 lb) of fuel burned (USDA Forest Service, 1976).

(4) All herbicide residue remaining on a treated site is released to the atmosphere at the time of burning.

Based on the above assumptions, the estimated volume of smoke produced is: (50,000 mg/kg) x (1 m^3/5mg) x 17,800 kg/ha = 30,000,000 m^3/ha.

The minimum and typical time intervals between herbicide application and burning for herbicides used in brown-and-burn operations in Region 8 are given in table 4-22. Degradation rates (k) of the herbicides on vegetation were used to estimate the fraction of applied herbicide remaining at the time of burning (see table 4-23). A degradation rate was not available for limonene; therefore, a k of 0.0 was used to avoid underestimating exposures.

Table 4-22

The number of days between herbicide treatment and prescribed burning (range is for all methods of application)

Herbicide[a]	Minimum	Typical
2,4-D amine	30	60 - 165
2,4-D ester	30	<45
2,4-DP	30	45 - 60
Glyphosate	30	70 - 180
Hexazinone	30	45 - 119
Imazapyr	30	50 - 100
Limonene	30	45 - 180
Picloram	30	60 - 171
Sulfometuron methyl	30	65 - 105
Triclopyr amine	30	60 - 100
Triclopyr ester	30	60 - 180

[a] Dicamba, fosamine, light fuel oil, and tebuthiuron and are not used in brown and burn operations.

Table 4-23

Degradation rates (k) for herbicides on vegetation

Herbicide[a]	k[b]	Source
2,4-D amine	0.0431	USDA, 1984
2,4-D ester	0.0431	USDA, 1984
2,4-DP	0.0431	USDA, 1984
Glyphosate	0.0495	Newton and Dost, 1981
Hexazinone	0.0584	USDA, 1984
Imazapyr	0.0266	Michael, 1986
Limonene	No data	
Picloram	0.0693	Bovey et al., 1967
Sulfometuron methyl	< 0.347	Michael and Neary, 1987
Triclopyr amine	0.022	Newton et al., 1982
Triclopyr ester	0.0040	Newton et al., 1982

[a] Dicamba, fosamine, light fuel oil, and tebuthiuron and are not used in brown and burn operations.

[b] k = fraction of remaining herbicide residue that is degraded per day.

Maximum potential exposures were calculated assuming a wildfire occurred in an area the day of herbicide treatment. The exposures were calculated in the same way as the exposures for the minimum and typical time intervals. It was assumed no herbicide degradation occurred.

An example calculation follows for determining the concentration in smoke of an herbicide; in this case hexazinone typical-aerial:

- Convert the application rate in lb/acre to mg/ha:

  $$1.7 \text{ lb/ac} \times (4.54 \times 10^5 \text{ mg/lb}) \times (2.5 \text{ ac/ha}) = 1{,}900{,}000 \text{ mg/ha}$$

- Determine the fraction of initial herbicide residue remaining at the time of burning. The degradation rate $k = 0.0584$ (table 4-22) and $t = 50$ days:

  $$\text{Fraction} = e^{-kt} = e^{-(0.0584 \times 50)} = 0.054$$

- The residue released at the time of burning is:

  $$1{,}900{,}000 \text{ mg/ha} \times 0.054 = 100{,}000 \text{ mg/ha}$$

- Using the estimated volume of smoke produced per ha, the concentration in the smoke is:

  $$(100{,}000 \text{ mg/ha}) \,/\, (30{,}000{,}000 \text{ m}^3\text{/ha}) = 0.0033 \text{ mg/m}^3$$

Worker Exposure and Dose Estimation

Doses for each worker category were estimated by extrapolating from the average dose levels found in field studies of workers exposed to 2,4-D using the same or a similar application method. Field studies of the exposures and resultant doses of workers using a variety of application equipment have been conducted on 2,4-D by Lavy et al. (1982, 1984), Nash et al. (1982), and Franklin et al. (1982). Doses for each worker category found in the studies are listed in table 4-24.

Lavy et al. (1982) monitored three helicopter spray crews for worker exposure to 2,4-D, using portable air filters, denim patches, and urine analysis on two separate spraying dates; the first observing normal precautions, the second using special protective clothing and procedures. Nash et al. (1982) monitored exposure of workers to 2,4-D during aerial spraying in Washington and ground spraying in North Dakota under normal spray conditions (that is, without special precautions). Lavy et al. (1984) investigated herbicide exposure to four spraying crews of 20 workers each, monitoring urine levels over two 5-day periods. Franklin et al. (1982) estimated worker exposure in pasture brush clearing operations in Saskatchewan using techniques similar to Lavy and coworkers. Urine samples were collected from personnel who conducted operations on 3 of 4 consecutive days.

The following steps were involved in estimating the worker doses from the field study data:

Table 4-24

Doses of 2,4-D measured in exposure studies for each worker category

Investigator	Application Type and Rule	Equipment Used	Worker Category	Number of Workers	Method of Analysis	Doses (mg/kg/day)	
						Average	Range
Lavy et al., 1982	Aerial, 2.2 kg a.i/ha	Helicopter	Flagman	2			0.00119–0.00177
			Pilot	3	Denim Patches	0.00057	n.d.–0.0010
			Mechanic	3		0.0233	0.0233–0.0617
			Batchman	3		0.0448	0.0233–0.0911
			Supervisor	3		0.0167	n.d.–0.0005
			Observer	6		8×10^{-5}	n.d.–0.0005
			Pilot	3	Urine	0.00248	0.00179–0.0557
			Mechanic	3		0.00068	0.00044–0.0136
			Batchman	3		0.00245	0.00215–0.0377
			Supervisor	3		0.00029	n.d.–0.0069
			Observer	6		0.00006	n.d.–0.0013
	Aerial	Helicopter, Special Precautions: Protective Coveralls, Gloves, Boots Hats, Goggles	Pilot	3	Denim Patches	3.3×10^{-5}	n.d.–0.0001
			Mechanic	3		0.00577	0.0005–0.0162
			Batchman	3		0.01065	0.00016–0.0216
			Supervisor	3		0.0009	n.d.–0.0027
			Observer	6		0.0014	n.d.–0.0045
			Pilot	3	Urine	0.00854	n.d.–0.0237
			Mechanic	3		0.00301	n.d.–0.00516
			Batchman	3		0.01401	0.00053–0.0219
			Supervisor	3		0.00013	n.d.–0.00038
			Observer	6		9 x 10-5	n.d.–0.00056
Nash et al., 1982	Aerial 585 kg a.i. applied in 20 hrs	4 Thrush Commanders 4 Grumman Ag-Cats 4 Pipers 1 Snow 1 Cessna	Mixer-loader	6	Urine	0.0199	0.0008–0.0545
			Mixer/loader-pilot	1		0.0180	
	360 kg a.i. applied in 14 hrs		Pilot	10		0.006	0.0013–0.0202

Table 4-24 (continued)

Doses of 2,4-D measured in exposure studies for each worker category

Investigator	Application Type and Rule	Equipment Used	Worker Category	Number of Workers	Method of Analysis	Doses (mg/kg/day)	
						Average	Range
	Ground 34 kg a.i. applied in 3.5 hrs.	Sprayers: 4 Pull-type 21 Self-propelled 10 cab 16 no cab	Sprayers	9	Urine	0.012	n.d.-0.0760
	18 kg a.i. applied in 2.4 hrs.		Mixer-loaded	7		0.0068	0.00165-0.0164
	38 kg a.i. applied in 7.9 hrs.		Mixer/loader-sprayer	8		0.020	0.0037-0.0442
Franklin et al., 1982	Aerial, 1.68-2.24 kg a.i./ha	Helicopter	Pilot Mixer	1 1	Urine	0.00322 0.013	
Lavy et al., 1984 (As cited in USDA, 1984)	Ground Weedone 170 (50% 2.3-D 50% 2.4-DP) 1 gal. herbicide/24 gal. water	Backpack	Operator	20	Urine	0.01752	n.d.-0.0903
	Tordon 101-R (80% 2,4-D, 20% Picloram)	Injection bar	Operator	20	Urine	0.0019	n.d.-0.0095
	Tordon 101-R	Hypohatchet	Operator	20	Urine	0.01696	n.d.-0.0866
	Tordon 101-R	Hack and squirt	Operator	20	Urine	0.00576	n.d.-0.0451
Note: protective coveralls, gloves, boots hats, goggles used	Weedon 170	Backpack	Operator	20	Urine	0.0196	0.0004-0.117
	Tordon 101-R	Injection bar	Operator	20		0.00086	n.d.-0.0035
	Tordon 101-R	Hypohatchet	Operator	20		0.0079	n.d.-0.0439
	Tordon 101-R	Hack and squirt	Operator	20		0.00244	n.d.-0.0148
	Tordon 101-R		Operator	20			

(1) The average dose observed in the 2,4-D field study is expressed in terms of dose per mass of active ingredient applied.

(2) The amount of active ingredient expected to be applied in the typical case was estimated by multiplying the herbicide's typical application rate by the typical area treated per day (from table 4-3).

(3) The amount of active ingredient expected to be applied in the maximum case was estimated by multiplying the herbicides's maximum application rate by the maximum area treated per day. In some cases, the maximum area treated at one time (table 4-3) is more than would normally be treated on a single day, so a smaller area was assumed to be treated per day.

(4) The herbicide-specific dose was estimated by multiplying the amount of herbicide applied by the dose of 2,4-D per unit of 2,4-D applied for that worker category in the field studies and then adjusting for the herbicide's dermal penetration rate.

(5) Typical exposures were adjusted if protective clothing is required by the label. The adjustment factors are discussed in section 5. Maximum exposures assume no protective clothing is worn, although this violates label warnings and restrictions.

Doses to workers applying solutions using hand application methods were estimated based on the hack-and-squirt method studied by Lavy. In these cases, the amount of active ingredient applied was not calculated on an area basis, but it was assumed to be proportional to the hours worked and the concentration of the herbicide formulation. This calculation was used for cut surface, basal bark/stem, and basal soil or soil spot treatments.

The methods used here for worker exposure estimation are likely to overestimate exposure in many cases. For example, the techniques used in Region 8 for basal soil, soil spot, and basal bark/stem applications probably involve significantly less contact with the herbicide solution than was the case in the Lavy field study. Also, the backpack sprayers in the Lavy study were treating vegetation that is much taller and more dense than typically treated in Region 8. However, because no field studies have been identified using methods equivalent to those used by the Forest Service in Region 8, a conservative approach has been taken to avoid underestimating risks.

It should also be noted that during the field exposure studies, many of the less severe types of accidents occurred that could be termed operational errors. For example, pilots handled the transfer hoses and helped with the mixing and loading operations and, in one instance when a pump broke down, transferred spray mix by bucket to the spray tank. In both of these cases, these individuals received higher doses during that day's work than they would have otherwise. Nevertheless, their doses were used in deriving the average worker doses for that field study.

Estimation of Doses to Workers and the Public From Accidents

The following scenarios were used to estimate the maximum realistic doses that would result from exposure to high amounts of herbicide in accidents.

(1) Accidental Spraying. Members of the public are accidentally sprayed with herbicide because they are beneath a spray aircraft or too close to a truck or backpack applicator. (This dose would also apply to workers.)

(2) Spills. (a) Members of the public receive herbicide exposure via drinking water when a load of herbicide mixture is spilled or when a container of herbicide concentrate breaks open and spills into a drinking water supply. (b) Workers spill concentrate on their skin during mixing or loading operations or are doused when a transfer hose breaks.

Accidental dermal cases were derived from modeling the dermal penetration of herbicide concentrate for direct exposures. Accidental ingestion doses were estimated by modeling the dilution of herbicide concentrate or mixture in a body of water of a given size.

To calculate the dose to a person directly sprayed at the full per-acre application rate, the maximum application rates were converted to mg/m^2. It was assumed that 0.186 m^2 of a person's skin is exposed.

An individual receives an accidental ingestion exposure resulting from a major spill by drinking water from a pond or a reservoir that has been contaminated by a dump of 379 l of herbicide mix as from a helicopter. This amount is approximately the largest load that can be carried by the types of helicopters to be used by the Forest Service. The pond is assumed to be 0.405 ha in area and 1.2 m deep, and to have no inflow or outflow. The reservoir is assumed to be 6.48 ha in area and 2.44 m deep. A person is assumed to drink 1 liter of water after complete mixing has occurred.

Direct dermal exposures were calculated for spills of 0.5 l of herbicide concentrate (if liquid concentrates are used). The person exposed during the spill is assumed to weigh 50 kg, and most of his surface area (0.8 m^2) is thoroughly wetted by the solution. Denim fabric commonly used in clothing retains about 57.5 ml of solution per square foot (Weeks, 1985), and adsorption of herbicide through the cloth was calculated as before, based on Newton and Norris (1981). However, 20 percent of the solution was assumed to soak bare skin. A spill resulting in this exposure could result from broken hoses or spilled containers.

Effect of Body Size on Exposure

All doses estimated in the exposure analysis were calculated for a representative 50-kg person. This weight was chosen to represent an adult of less than average weight, so that doses to adults would be calculated in a conservative manner. Doses for a larger person would be less in terms of mg per kg body weight. For example, a 70-kg person would receive approximately 25 percent more herbicide than a 50-kg person by dermal exposure because of his greater surface area. A 70-kg person would also receive on average about 25 percent more herbicide by dietary exposure routes because both body surface area and metabolism are approximately proportional to body weight raised to the 2/3 power:

$$\frac{(70)^{2/3}}{(50)^{2/3}} = 1.25$$

However, a 70-kg person also has a body weight greater than a 50-kg person, by a greater factor:

$$\frac{70}{50} = 1.4$$

The combined effect of these two factors is that a 70-kg person will receive a dose in mg/kg that is only 89 percent as great as for a 50-kg person.

Conversely, smaller people can be expected to receive greater doses in terms of mg per kg body weight. A 20-kg child will receive only about 54 percent as much herbicide as a 50-kg person, but his weight is only 40 percent as great. The net effect is that a 20-kg child will receive a dose that is 36 percent greater in terms of mg/kg than it would be for a 50-kg person.

Estimation of Lifetime Doses to Workers and the Public

Doses used in the cancer risk analysis for 2,4-D, 2,4-DP, picloram, glyphosate, and light fuel oil (discussed in section 5) were derived by combining the available information on the number of days per year an individual worker may use an herbicide with a particular application method and estimates of the expected daily dose and the number of years of employment. The number of years of employment was taken to be 20 in all cases. The doses were calculated assuming that the maximum application rates and acreage were encountered 1 year in 10, and typical rates and acreage 9 years in 10.

Lifetime exposures to the public were calculated assuming 10 typical or 10 maximum exposures.

REPRESENTATIVE EXPOSURES

The representative exposures are presented in tables 4-25 through 4-40 for each of the chemicals. These exposures will be used for calculation of margins of safety, which are presented in section 5.

Table 4-25

2,4-D amine exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00012	0.00028
Onsite	0.00090	0.00144
Dietary		
Water	0.00006	0.00014
Fish	0.00001	0.00003
Meat	0.00004	0.00568
Vegetable	0.00048	0.00107
Berry Picking	0.00040	0.04659
Workers		
Aerial		
Pilot	0.00633	0.04832
Mixer/Loader	0.01575	0.06912
Observer	0.00021	0.00157
Mechanical Ground		
Applicator	0.00535	0.06857
Mixer/Loader	0.01273	0.06985
Appl/Mix/Load	0.01254	0.09639
Manual Ground		
Backpack	0.06173	0.44514
Cut Surface	0.02523	0.07933
Accidents		
Spill Onto Worker		136.8000
Accidental Spray		0.1001
Spills Into Water		
Ground--18.9 l Into Pond		0.0350
Air--379 l Into Reservoir		0.0023

Table 4-26

2,4-D ester exposures

Category	Exposure Typical (mg/kg/day)	 Maximum
Public		
Dermal		
Drift	0.00019	0.00049
Onsite	0.00144	0.00251
Dietary		
Water	0.00010	0.00025
Fish	0.00002	0.00005
Meat	0.00007	0.00994
Vegetable	0.00077	0.00187
Berry Picking	0.00064	0.08154
Workers		
Aerial		
Pilot	0.00791	0.07248
Mixer/Loader	0.01968	0.10368
Observer	0.00026	0.00235
Mechanical Ground		
Applicator	0.00642	0.09600
Mixer/Loader	0.01528	0.09778
Appl/Mix/Load	0.01505	0.13494
Manual Ground		
Backpack	0.06173	0.59351
Basal Stem	0.00984	0.02320
Accidents		
Spill Onto Worker		136.8000
Accidental Spray		0.1502
Spills Into Water		
Ground--18.9 l Into Pond		0.0350
Air--379 l Into Reservoir		0.0035

Table 4-27

2,4-DP exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00000	0.00001
Onsite	0.00002	0.00004
Dietary		
Water	0.00010	0.00022
Fish	0.00002	0.00004
Meat	0.00006	0.00717
Vegetable	0.00077	0.00160
Berry Picking	0.00064	0.06989
Workers		
Aerial		
Pilot	0.00016	0.00063
Mixer/Loader	0.00039	0.00090
Observer	0.00001	0.00002
Mechanical Ground		
Applicator	0.00014	0.00137
Mixer/Loader	0.00034	0.00140
Appl/Mix/Load	0.00033	0.00193
Manual Ground		
Backpack	0.00051	0.00742
Basal Stem	0.00016	0.00063
Accidents		
Spill Onto Worker		2.2200
Accidental Spray		0.0021
Spills Into Water		
Ground--18.9 l Into Pond		0.0341
Air--379 l Into Reservoir		0.0029

Table 4-28

Dicamba exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00011	0.00024
Onsite	0.00083	0.00125
Dietary		
Water	0.00005	0.00011
Fish	0.00001	0.00002
Meat	0.00003	0.00437
Vegetable	0.00039	0.00080
Berry Picking	0.00032	0.03494
Workers		
Mechanical Ground		
Applicator	0.00496	0.05966
Mixer/Loader	0.01181	0.06077
Appl/Mix/Load	0.01164	0.08386
Manual Ground		
Cut Surface	0.00770	0.03027
Accidents		
Spill Onto Worker		41.7600
Accidental Spray		0.0871
Spills Into Water		
Ground--18.9 l Into Pond		0.0092
Air--379 l Into Reservoir		----

Table 4-29

Diesel exposures

Category	Exposure Typical (mg/kg/day)	 Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00039	0.00101
Onsite	0.00299	0.00524
Dietary		
Water	0.00005	0.00013
Fish	0.00005	0.00013
Meat	0.00004	0.00751
Vegetable	0.00039	0.00093
Berry Picking	0.00032	0.04077
Workers		
Aerial		
Pilot	0.00395	0.04719
Mixer/Loader	0.00984	0.06750
Observer	0.00013	0.00153
Mechanical Ground		
Applicator	0.02674	0.25000
Mixer/Loader	0.06365	0.25465
Appl/Mix/Load	0.06271	0.35141
Manual Ground		
Basal Stem	0.07336	0.23068
Accidents		
Spill Onto Worker		1020.0000
Accidental Spray		0.1564
Spills Into Water		
Ground--18.9 l Into Pond		0.0626
Air--379 l Into Reservoir		0.0009

Table 4-30

Fosamine exposures

Category	Exposure Typical (mg/kg/day)	 Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00061	0.00139
Onsite	0.00464	0.00718
Dietary		
Water	0.00020	0.00043
Fish	0.00004	0.00009
Meat	0.00013	0.01886
Vegetable	0.00150	0.00320
Berry Picking	0.00124	0.13978
Workers		
Aerial		
Pilot	0.03163	0.20133
Mixer/Loader	0.07873	0.28800
Observer	0.00103	0.00653
Mechanical Ground		
Applicator	0.00691	0.34286
Mixer/Loader	0.01644	0.34923
Appl/Mix/Load	0.01620	0.48193
Manual Ground		
Backpack	0.20575	1.97838
Accidents		
Spill Onto Worker		240.0000
Accidental Spray		0.4171
Spills Into Water		
Ground--18.9 1 Into Pond		0.0368
Air--379 1 Into Reservoir		0.0058

Table 4-31

Glyphosate exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00012	0.00046
Onsite	0.00090	0.00239
Dietary		
Water	0.00004	0.00014
Fish	0.00001	0.00003
Meat	0.00003	0.00629
Vegetable	0.00029	0.00107
Berry Picking	0.00024	0.04659
Workers		
Aerial		
Pilot	0.00237	0.08053
Mixer/Loader	0.00590	0.11520
Observer	0.00008	0.00261
Mechanical Ground		
Applicator	0.00401	0.45714
Mixer/Loader	0.00955	0.46564
Appl/Mix/Load	0.00941	0.64257
Manual Ground		
Backpack	0.05144	0.98919
Cut Surface	0.03319	0.10438
Accidents		
Spill Onto Worker		180.0000
Accidental Spray		0.1668
Spills Into Water		
Ground--18.9 l Into Pond		0.0276
Air--379 l Into Reservoir		0.0023

Table 4-32

Hexazinone exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00013	0.00069
Onsite	0.00102	0.00359
Dietary		
Water	0.00004	0.00022
Fish	0.00002	0.00009
Meat	0.00003	0.00943
Vegetable	0.00033	0.00160
Berry Picking	0.00027	0.06989
Workers		
Aerial		
Pilot	0.00791	0.06040
Mixer/Loader	0.01968	0.08640
Observer	0.00026	0.00196
Mechanical Ground		
Applicator	0.00833	0.85714
Mixer/Loader	0.01984	0.87308
Appl/Mix/Load	0.01954	1.20482
Manual Ground		
Backpack	0.01029	0.98919
Soil Spot	0.01106	0.03479
Accidents		
Spill Onto Worker		120.0000
Accidental Spray		0.1251
Spills Into Water		
Ground--18.9 l Into Pond		0.0184
Air--379 l Into Reservoir		0.0017

Table 4-33

Imazapyr exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00006	0.00017
Onsite	0.00045	0.00090
Dietary		
Water	0.00002	0.00005
Fish	0.00000	0.00001
Meat	0.00001	0.00236
Vegetable	0.00014	0.00040
Berry Picking	0.00012	0.01747
Workers		
Aerial		
Pilot	0.00237	0.03020
Mixer/Loader	0.00590	0.04320
Observer	0.00008	0.00098
Mechanical Ground		
Applicator	0.00368	0.21429
Mixer/Loader	0.00875	0.21827
Appl/Mix/Load	0.00862	0.30120
Manual Ground		
Backpack	0.05144	0.49459
Cut Surface	0.02213	0.06089
Accidents		
Spill Onto Worker		120.0000
Accidental Spray		0.0626
Spills Into Water		
Ground--18.9 l Into Pond		0.0184
Air--379 l Into Reservoir		0.0009

Table 4-34

Kerosene exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00044	0.00131
Onsite	0.00340	0.00679
Dietary		
Water	0.00006	0.00016
Fish	0.00006	0.00016
Meat	0.00004	0.00974
Vegetable	0.00044	0.00121
Berry Picking	0.00036	0.05288
Workers		
Aerial		
Pilot	0.01496	0.22851
Mixer/Loader	0.03723	0.32688
Observer	0.00049	0.00742
Mechanical Ground		
Applicator	0.02529	2.02679
Mixer/Loader	0.06020	2.06446
Appl/Mix/Load	0.05931	2.84890
Manual Ground		
Backpack	0.07304	1.05101
Basal Stem	0.02449	0.07701
Accidents		
Spill Onto Worker		340.5000
Accidental Spray		0.4734
Spills Into Water		
Ground--18.9 l Into Pond		0.0209
Air--379 l Into Reservoir		0.0026

Table 4-35

Limonene exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00007	0.00042
Onsite	0.00054	0.00215
Dietary		
Water	0.00002	0.00013
Fish	0.00000	0.00003
Meat	0.00002	0.00566
Vegetable	0.00017	0.00096
Berry Picking	0.00014	0.04193
Workers		
Aerial		
Pilot	0.00285	0.07248
Mixer/Loader	0.00709	0.10368
Observer	0.00009	0.00235
Mechanical Ground		
Applicator	0.00401	0.64286
Mixer/Loader	0.00955	0.65481
Appl/Mix/Load	0.00941	0.90361
Manual Ground		
Backpack	0.04629	0.44514
Basal Stem	0.03107	0.09770
Accidents		
Spill Onto Worker		432.0000
Accidental Spray		0.1502
Spills Into Water		
Ground--18.9 l Into Pond		0.0663
Air--379 l Into Reservoir		0.0021

Table 4-36

Picloram exposures

Category	Exposure Typical	 Maximum
	(mg/kg/day)	
Public		
Dermal		
Drift	0.00000	0.00000
Onsite	0.00001	0.00002
Dietary		
Water	0.00002	0.00005
Fish	0.00000	0.00001
Meat	0.00001	0.00168
Vegetable	0.00014	0.00037
Berry Picking	0.00011	0.01631
Workers		
Aerial		
Pilot	0.00005	0.00054
Mixer/Loader	0.00012	0.00078
Observer	0.00000	0.00002
Mechanical Ground		
Applicator	0.00004	0.00072
Mixer/Loader	0.00011	0.00073
Appl/Mix/Load	0.00011	0.00101
Manual Ground		
Backpack	0.00037	0.00223
Cut Surface	0.00006	0.00021
Accidents		
Spill Onto Worker		0.2916
Accidental Spray		0.0011
Spills Into Water		
Ground--18.9 l Into Pond		0.0025
Air--379 l Into Reservoir		0.0009

Table 4-37

Sulfometuron methyl exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00001	0.00004
Onsite	0.00010	0.00022
Dietary		
Water	0.00000	0.00001
Fish	0.00000	0.00000
Meat	0.00000	0.00058
Vegetable	0.00003	0.00010
Berry Picking	0.00003	0.00431
Workers		
Aerial		
Pilot	0.00069	0.00805
Mixer/Loader	0.00171	0.01152
Observer	0.00002	0.00026
Mechanical Ground		
Applicator	0.00050	0.00846
Mixer/Loader	0.00119	0.00861
Appl/Mix/Load	0.00117	0.01189
Manual Ground		
Backpack	0.00309	0.06182
Accidents		
Spill Onto Worker		210.0000
Accidental Spray		0.0167
Spills Into Water		
Ground--18.9 l Into Pond		0.0322
Air--379 l Into Reservoir		0.0002

Table 4-38

Tebuthiuron exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00001	0.00018
Onsite	0.00060	0.00359
Dietary		
Water	0.00001	0.00009
Fish	0.00002	0.00019
Meat	0.00001	0.00943
Vegetable	0.00005	0.00052
Berry Picking	0.00006	0.06989
Workers		
Aerial		
Pilot	0.00633	0.12080
Mixer/Loader	0.01575	0.17280
Observer	0.00021	0.00392
Mechanical Ground		
Applicator	0.00357	0.17143
Mixer/Loader	0.00849	0.17462
Appl/Mix/Load	0.00836	0.24096
Manual Ground		
Backpack	0.20575	1.73108
Soil Spot	0.00039	0.00148
Accidents		
Spill Onto Worker		5.1000
Accidental Spray		0.2503
Spills Into Water		
Ground--18.9 l Into Pond		0.0782
Air--379 l Into Reservoir		0.0035

Table 4-39

Triclopyr amine exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00005	0.00015
Onsite	0.00039	0.00079
Dietary		
Water	0.00010	0.00029
Fish	0.00002	0.00006
Meat	0.00006	0.01003
Vegetable	0.00077	0.00214
Berry Picking	0.00064	0.09318
Workers		
Aerial		
Pilot	0.00261	0.01993
Mixer/Loader	0.00650	0.02851
Observer	0.00008	0.00065
Mechanical Ground		
Applicator	0.00588	0.18857
Mixer/Loader	0.01400	0.19208
Appl/Mix/Load	0.01380	0.26506
Manual Ground		
Backpack	0.01188	0.08161
Cut Surface	0.00456	0.01938
Accidents		
Spill Onto Worker		29.7000
Accidental Spray		0.0413
Spills Into Water		
Ground--18.9 L Into Pond		0.0276
Air--379 L Into Reservior		0.0035

Table 4-40

Triclopyr ester exposures

Category	Exposure Typical (mg/kg/day)	Exposure Maximum (mg/kg/day)
Public		
Dermal		
Drift	0.00005	0.00015
Onsite	0.00039	0.00079
Dietary		
Water	0.00010	0.00029
Fish	0.00002	0.00006
Meat	0.00006	0.01003
Vegetable	0.00077	0.00214
Berry Picking	0.00064	0.09318
Workers		
Aerial		
Pilot	0.00174	0.02658
Mixer/Loader	0.00433	0.03802
Observer	0.00006	0.00086
Mechanical Ground		
Applicator	0.00294	0.23571
Mixer/Loader	0.00700	0.24010
Appl/Mix/Load	0.00690	0.33133
Manual Ground		
Backpack	0.00849	0.12241
Basal Stem	0.00285	0.00896
Accidents		
Spill Onto Worker		39.6000
Accidental Spray		0.0551
Spills Into Water		
Ground--18.9 L Into Pond		0.0368
Air--379 L Into Reservoir		0.0046

Section 5

HUMAN HEALTH RISK ANALYSIS

This section discusses the potential risks to the health of workers and members of the public from the Forest Service's proposed herbicide applications in Region 8 by comparing the exposure levels estimated in section 4 with the toxic effect levels described in section 3. The first subsection describes the methods used to evaluate human health risks. The second subsection summarizes results of the human health risk analysis. The third subsection evaluates the risks of threshold effects, which include acute toxic effects, chronic systemic effects, and reproductive (fetotoxic, maternal toxic, and teratogenic) effects, including any other effects on reproductive success. Public risks of effects from typical and maximum exposures from routine operations and from accidental exposures are considered first in this subsection. Risks from brown-and-burn operations also are discussed. In addition, the probability of occurrence of these various exposures are considered. Risks to workers from typical and maximum exposures and from accidents are discussed next. The subsection also contains a discussion of the influence of protective clothing on worker exposures. The fourth subsection evaluates the risks of the herbicides causing cancer and the fifth, the risk of the chemicals causing heritable mutations. The final subsection discusses the risks of synergistic effects, cumulative effects, and effects on sensitive individuals. All judgments about risk consider the possible exposure levels and the likelihood that the estimated exposures would actually occur.

HOW RISKS TO WORKERS AND THE PUBLIC WERE DETERMINED

In this analysis, the risks to humans exposed to the 11 herbicides and 3 associated chemicals were quantified by comparing the doses estimated in the range of exposure scenarios presented in section 4 with the results of toxicity tests on laboratory animals described in section 3. In essence, the risks of threshold effects are quantified by dividing a laboratory NOEL by an estimated dose to produce a margin of safety (MOS).

The Margin of Safety for Threshold Effects

There are two basic approaches for extrapolating from laboratory animal NOEL's to the general human population: the acceptable daily intake (ADI) approach using specified "safety factors" and the margin of safety approach. Using the acceptable daily intake approach, safety factors based on the quality of the data are applied to the highest dose that produces no effects in animal studies for the estimation of acceptable human daily exposures (Thomas, 1986; Klaassen and Doull, 1980). (EPA currently uses the term Reference Dose, or RFD, when referring to the acceptable daily intake. ADI and RFD are synonymous.) An uncertainty factor of 10 has normally been used for estimating safe levels for humans when there are valid human studies available and no indication of carcinogenicity. An uncertainty factor of 100 has been used when there are few or no human studies available but there are valid long-term animal studies. When toxicological data are limited, a factor of 1,000 or greater might be used to estimate acceptable human exposure. For example, EPA used a safety factor of 2,000 to set the ADI for dicamba because the results of a subchronic study were used.

Safety factors and the "ADI approach" are used by Federal regulatory agencies, such as the Food and Drug Administration (FDA) and EPA, to set ADI's for chemicals that a broad segment of the general public are likely to be exposed to for an indeterminate period of time. Thus, the ADI is considered a lifetime safe dose for threshold toxic effects, based on the best available toxicity information on a particular chemical. The ADI is not anticipated to result in any adverse effects after chronic exposure to the general population of humans, including sensitive subgroups (Dourson and Stara, 1983). Cancer and heritable mutation effects are not dealt with in this way because they are not assumed to have a predictable threshold of reversible toxic effects. In brief, the ADI approach begins with a NOEL and safety factor to develop a safe dose estimate.

The MOS approach, on the other hand, although it is based on the same concepts of a threshold of toxicity (approximated by animal NOEL's in long-term studies) and of the safety of a dose, begins with a NOEL and an estimated human dose to develop an index of risk--the margin of safety. This method differs from the ADI approach in several important ways. First, the MOS approach is not being used here to establish a regulatory standard safe level for the general public against which samples of possibly contaminated products, such as marketed vegetables or drinking water, would be tested. The MOS's computed here are NOEL:dose ratios that are direct comparisons of the doses estimated in this risk assessment with the NOEL's from animal studies. For example, an MOS of 100 means the NOEL is 100 times higher than the estimated dose. Although the MOS's correspond with the safety factors used to determine the ADI's, they are used for comparative purposes and are applicable only to this risk assessment. Also, a margin of safety does not always mean that the dose is safe. An MOS of 3, for example, represents a risk of toxic effects for repeated exposures.

Second, the ADI, as a standard level for comparing tested samples, should remain relatively stable over the years, modified only if the results of further toxicity tests produce a new NOEL or make a change in the ADI safety factor appropriate. The margins of safety in this risk assessment, however, vary with the estimated doses in a particular exposure scenario. The MOS's are used to indicate the potential toxic effects of a proposed herbicide under differing conditions or routes of exposure or in comparison with alternative herbicides that may be used for the same purpose.

The larger the margin of safety (the smaller the estimated human dose compared to the animal NOEL), the lower the risk to human health. As the estimated dose to humans approaches the animal NOEL (as the MOS approaches one), the risk to humans increases. When an estimated dose exceeds a NOEL (giving an MOS of less than one), the ratio is reversed (the dose is divided by the NOEL) to indicate how high the estimated dose is above the laboratory toxicity level; a minus sign is attached to indicate that the dose exceeded the NOEL; and the result is no longer termed a margin of safety but is called simply a negative ratio.

A ratio of -3, for example, means that the estimated dose is 3 times the NOEL. A negative ratio indicates that the estimated dose (given all the assumptions of the scenario) represents a clear risk of toxic effects for repeated doses and some possibility of acute effects for doses that are likely to occur only rarely. Comparing one-time or once-a-year doses (such as those that may be experienced by the public) to NOEL's derived from lifetime studies tends to overestimate the risk from those rare events.

In general, for those chemicals with valid long-term toxicity studies, when repeated doses to humans approach the animal NOEL (the MOS is less than 10), there is a clear possibility of harmful effects. When the MOS is less than 100, some members of the public, particularly sensitive individuals, may be at risk. Conversely, when the human dose is small compared with the animal NOEL (giving an MOS greater than 100), the risk to the general public can be judged negligible, including most sensitive individuals.

For doses that are not likely to occur more than once, such as the dose a worker receives from spilling a half liter of spray mix over his entire upper body, an estimated dose that exceeds the laboratory test animal NOEL does not necessarily lead to the conclusion that there will be toxic effects. In fact, because all of the NOEL's used in this risk analysis are based on (or take into account) long-term exposure, the dose would likely have to far exceed the NOEL to cause toxic effects. Estimated doses of this kind that exceed the NOEL also are compared to the herbicide's acute oral LD_{50}, so that a more realistic judgment can be made about the risk of acute toxic effects, including death.

Systemic effects are evaluated based on the lowest systemic NOEL found in a 2-year feeding study of dogs, rats, or mice. When only subchronic studies were available, or if subchronic studies reported effects at lower levels than chronic studies, the subchronic NOEL's were used (for example, for triclopyr. See section 3 for details). Reproductive effects are evaluated based on the lowest maternal or fetotoxic NOEL found in a three-generation reproductive study or the lowest teratogenic NOEL found in a teratology study. The lowest NOEL, either systemic or reproductive, for each chemical was used to compute the MOS's. LD_{50}'s and systemic and reproductive NOEL's used in the risk analysis are listed in table 5-1.

Systemic NOEL's were not available for kerosene and diesel oil, so NOEL's were estimated as the LD_{50} divided by 1,000. This factor of 1,000 is exceeded by only one of the other 12 chemicals studied. The factor is less than 400 for all of the other chemicals. Thus, although this is a source of uncertainty in the analysis, the NOEL's for diesel oil and kerosene were chosen in a manner that is intended to be conservative.

Analysis of Nonthreshold Risks

An analysis of cancer risk was conducted for the herbicides for which there are positive cancer studies or for which there is scientific controversy about its ability to cause cancer (2,4-D, 2,4-DP, picloram and glyphosate) and for the petroleum distillates kerosene and diesel oil that contain the known carcinogenic compounds benzene and benzo-a-pyrene. The risk of cancer is calculated for an individual by comparing estimates of lifetime

Table 5-1

Toxicity reference values

Chemical	Rat Oral LD$_{50}$ mg/kg	Systemic NOEL mg/kg/day	Reproductive NOEL[f] mg/kg/day	Human Cancer Potency[g]
2,4-D[a]	375	1	5	0.0262
2,4-DP	532	5	6.25	0.0648
Dicamba	757	15.8	3.0	---[c]
Diesel	7,380	7.38[b]	751	0.0000049
Fosamine	24,400	25	50	---[c]
Glyphosate	4,320	31	10	0.000323
Hexazinone	1,690	10	50	---[c]
Imazapyr	5,000[e]	500	300	---[c]
Kerosene	28,000[e]	28[b]	751[d]	0.0000049
Limonene	5,000	227	227	---[c]
Picloram	8,200	7	50	0.00296
Sulfometuron methyl	5,000[e]	2.5	25	---[c]
Tebuthiuron	644	12.5	5.0	---[c]
Triclopyr[a]	630	2.5	2.5[h]	---[c]

[a] Used for both amine and ester formulations.
[b] NOEL used in risk analysis based on LD$_{50}$: NOEL = LD$_{50}$/1,000.
[c] No oncogenic potential indicated in laboratory studies.
[d] Based on diesel oil NOEL.
[e] LD$_{50}$ level is "greater than" indicated dose.
[f] Refers to various kinds of effects observed in studies of reproduction and development. See section 3 for details.
[g] Upper 95 percent confidence limit, in units of per mg/kg/day.
[h] Based on systemic NOEL

dose over a 70-year period (computed in section 4), with cancer potency estimates derived in the Hazard Analysis section. The cancer potencies of these chemicals are listed in table 5-1.

An analysis is conducted for those herbicides that have positive mutagenicity tests or those for which no data are available. The risk of these herbicides causing mutations is qualitative rather than quantitative, with a statement of the probable risk based on the available evidence of mutagenicity and carcinogenicity.

SUMMARY OF THE HUMAN HEALTH RISK ANALYSIS

Public Risks

Comparison of estimated typical public exposures with laboratory toxicity levels indicates that no member of the public, including sensitive individuals, should be affected by the herbicides or associated chemicals used for vegetation management in Region 8. The lowest MOS (table 5-2) is 700 for 2,4-D systemic effects. All others are 1,000 or greater for both systemic and reproductive effects.

Public MOS's from maximum exposures (table 5-2) are low for 2,4-D amine (21) and ester (12) and triclopyr amine (27) and ester (27) for systemic effects. The MOS is also less than 100 for 2,4-DP systemic effects. The public may experience some toxic effects from these maximum exposures, but the effects should be short-lived and reversible. Triclopyr amine and ester also present a risk of reproductive effects. 2,4-D ester, 2,4-DP, and dicamba present somewhat lower reproductive effects risks. It is extremely unlikely that these effects would be experienced because the public is not likely to be exposed to these maximum doses more than a very limited number of times in their lifetime (table 5-3), if they are exposed at all. The public is at some risk from accidents for 10 of the 14 chemicals. 2,4-DP, imazapyr, limonene, and picloram do not present a risk to the public even in these situations. The normal operational safety precautions should limit the possibility of these exposures occurring.

Public cancer risks were found to be less than 1 in 1 million for all chemicals (table 5-26).

Worker Risks

Workers are at greater risk of systemic and reproductive effects (table 5-6) than members of the public. However, they are not at risk from typical or maximum exposures to 2,4-DP, imazapyr, limonene, or picloram. Workers are at risk of kidney effects from long-term typical exposures to 2,4-D and at risk of kidney effects from triclopyr typical exposures. None of the other chemicals present as high a risk as 2,4-D and triclopyr do from typical chronic exposures.

For workers, cancer risks are greater than 1 in 1 million for 2,4-D amine and ester mechanical and manual ground applications, and from 2,4-DP backpack applications. Diesel oil, glyphosate, kerosene, and picloram do not present a cancer risk of greater than 1 in 1 million to any type of worker.

Risk of Mutagenicity

None of the chemicals appears to present a significant risk of heritable mutations. Although there is no direct evidence for humans, the weight of evidence in mutagenicity assays indicates that all but 2,4-D, 2,4-DP, and the light fuel oils are not likely to affect DNA in human germ cells to produce heritable mutations. For the latter chemicals, it is reasonable to conclude that their mutagenic potential is weak.

Table 5-2

Lowest margins of safety for the public from typical and maximum estimated exposures in routine operations

Chemical	Typical Exposures		Maximum Exposures	
	Systemic	Reproductive	Systemic	Reproductive
2,4-D (amine)	1,100 (DEO)	5,570 (DEO)	21 (BP)	107 (BP)
2,4-D (ester)	700 (DEO)	3,481 (DEO)	12 (BP)	61 (BP)
2,4-DP	6,500 (VEG)	8,094 (VEG)	72 (BP)	89 (BP)
Dicamba	10,000	3,614 (DEO)	452 (BP)	86 (BP)
Diesel oil	2,500 (DEO)	10,000	180 (BP)	10,000
Fosamine	5,400 (DEO)	10,000	180 (BP)	358 (BP)
Glyphosate	10,000	10,000	670 (BP)	215 (BP)
Hexazinone	9,800 (DEO)	10,000	140 (BP)	715 (BP)
Imazapyr	10,000	10,000	10,000	10,000
Kerosene	8,200 (DEO)	10,000	530 (BP)	10,000
Limonene	10,000	10,000	5,400 (BP)	5,413 (BP)
Picloram	10,000	10,000	430 (BP)	3,066 (BP)
Sulfometuron methyl	10,000	10,000	580 (BP)	5,801 (BP)
Tebuthiuron	10,000	8,333	180 (BP)	72 (BP)
Triclopyr (amine)	1,000 (DEO)	1,045 (DEO)	27 (BP)	27 (BP)
Triclopyr (ester)	1,000 (DEO)	1,045 (DEO)	27 (BP)	27 (BP)

DEO: Dermal exposure - onsite.
VEG: Dietary - vegetables.
BP: Dietary - berry picking.

RISK OF GENERAL SYSTEMIC AND REPRODUCTIVE EFFECTS

Margins of safety were computed for each application scenario--routine-typical, routine-maximum, and accidents--for the public and workers for the 11 herbicides and 3 associated chemicals. The margins of safety were computed by comparing the laboratory-determined NOEL's and LD_{50}'s in table 5-1 with the doses computed in the exposure analysis.

Table 5-3

Margins of safety for the public exposed in accidents

Chemical	Direct Spray		Pond Spill		Reservoir Spill	
	Systemic	Repro.	Systemic	Repro.	Systemic	Repro.
2,4-D (amine)	10	50	29	143	430	2,172
2,4-D (ester)	7	33	29	143	290	1,448
2,4-DP	2,400	2,997	150	184	1,700	2,172
Dicamba	181	34	1,717	326	NA	NA
Diesel oil	47	4,802	120	10,000	8,600	10,000
Fosamine	60	120	680	1,359	4,300	8,621
Glyphosate	190	60	1,100	362	10,000	4,345
Hexazinone	80	399	540	2,716	5,800	10,000
Imazapyr	8,000	4,795	10,000	10,000	10,000	10,000
Kerosene	59	1,586	1,300	10,000	10,000	10,000
Limonene	3,000	3,024	3,400	3,425	10,000	10,000
Picloram	6,200	10,000	2,800	10,000	8,100	10,000
Sulfometuron methyl	150	1,499	78	776	10,000	10,000
Tebuthiuron	50	20	170	64	3,600	1,429
Triclopyr (amine)	61	61	91	91	720	724.1
Triclopyr (ester)	45	45	68	68	540	543

NA = not applicable.

Table 5-2 lists the lowest margins of safety for the public for the 14 herbicides and additives for the typical and maximum exposure scenarios. MOS's for both the amine and ester formulations of 2,4-D and triclopyr are listed because they are used at different rates or in different programs. Their toxicological differences are not significant. Table 5-3 summarizes MOS's for the public exposed in accidents. Table 5-4 concerns the likelihood of spill accidents. Table 5-5 indicates effects observed in lab animals at the lowest doses showing effects. These are the types of effects that may appear in humans when margins of safety are low if they are chronically exposed. Table 5-6 lists the lowest MOS's for workers for typical and maximum exposures. Table 5-7 gives MOS's and LD_{50} comparisons for worker spill accidents. The doses for the public and workers and the computed margins of safety for the individual chemicals are listed in tables 5-8 to 5-23.

Table 5-4

Number of spills on Forest Service land in Washington and Oregon over the last 10 years

Number of Gallons	Number Spills (air and ground)	Avg. No. of Spills/ 1,000 Acres	Number Spills (air)	Avg. No. of Spills/ 1,000 Acres
0-9	24	0.0795	9	0.0298
10-19	19	0.0629	9	0.0298
20-29	14	0.0464	6	0.0199
30-39	11	0.0364	5	0.0166
40-49	11	0.0364	5	0.0166
50-59	10	0.0331	4	0.0132
60-69	8	0.0265	4	0.0132
70-79	6	0.0199	2	0.0066
80-89	6	0.0199	2	0.0066
90-99	3	0.0099	2	0.0066
100 and more	1	0.0033	0	0.0000

Risk to the Public From Routine Operations

Risk to the Public From Typical Exposures

Margins of Safety for the Public for Typical Exposures. Table 5-2 shows that there are large margins of safety (1,000 or greater) for both systemic and reproductive effects for every category of typical exposure for every proposed herbicide except 2,4-D ester: it has a margin of safety of 700 for systemic effects. The lowest margins of safety are for dermal exposure onsite except for 2,4-DP (dietary-vegetable). Margins of safety for imazapyr, limonene, picloram, and sulfometuron methyl are greater than or equal to 10,000 for both systemic and reproductive effects. MOS's for dicamba, 10,000 for systemic effects and 3,614 for reproductive effects, exceed the safety factor of 2,000 used by EPA to set the acceptable daily intake for dicamba.

Although the public should not be chronically exposed to these herbicides (considering the remote location of most treated areas, it is unlikely that any member of the public will be exposed at all), these large margins of safety mean that members of the public could be repeatedly exposed to these

Table 5-5

Systemic effects of subchronic and chronic exposure to Region 8 herbicides observed in animals

Herbicide	Dose Level (mg/kg/day)	Effects Observed
2,4-D	5	Renal effects (increased tubular brown pigment and increased vacuolization of renal cortex cytoplasm) (EPA, 1986b); increased thyroid weight
	51.4	Stupor, incoordination, weak reflexes, urinary incontinence, human injection (USDA, 1984).
2,4-DP	25	Decreased blood sodium and packed blood cell volume; increased kidney and liver weight (EPA, 1984a)
	150	Decreased weight gain, decreased hematocrit and red blood cells, chronic prostatitis, and kidney degeneration (EPA, 1984a)
Dicamba	40	Slight liver cell necrosis and cytoplasmic vacuolization (EPA, 1984b)
Fosamine	125	Increased stomach weight (USDA, 1984)
Glyphosate	100	Decreased relative and absolute pituitary weight (EPA, 1986c)
Hexazinone	125	Decreased body weight and increased liver weight (EPA, 1986d)
	375	Increased liver size, localized increase in size and number of liver cells, and localized tissue degeneration (EPA, 1986d)
Imazapyr	500	No effects observed at highest dose tested (American Cyanamid, 1985)
Picloram	35	Increased liver weights (Mullison, 1985)
	60	Increased size and altered properties of liver cells (Dow, 1987a)
Sulfometuron methyl	250	Hemolytic effects, liver toxicity, and decreased mean absolute body and brain weights (DuPont, 1986)

Table 5-5 (continued)

Systemic effects of subchronic and chronic exposure
to Region 8 herbicides observed in animals

Herbicide	Dose Level (mg/kg/day)	Effects Observed
Tebuthiuron	25	Increased thyroid and spleen weight (EPA, 1986a) 90-day dog
	125	Growth suppression, pancreatic lesions (EPA, 1986a) 90-day rat
Triclopyr	5	Decreased weight gain and food consumption, liver and kidney effects due to increased urinary retention of triclopyr--183-day dog feeding (EPA, 1985)
	60	Decreased liver weight (USDA, 1984)--90-day mouse feeding
	100	Decreased body weight, food consumption, and absolute liver weights (EPA, 1985)--90-day rat feeding

typical levels and suffer no adverse effects. This is true for the general public, including pregnant women and the majority of sensitive individuals.

Risk to the public is low for all kinds of exposures, but they are especially low for direct exposure to drift and dietary exposures from fish, meat, and water. Risk is somewhat higher for dietary exposures from berries and vegetables, and dermal contact with vegetation onsite.

Probability of the Estimated Typical Public Exposures Occurring. Although the typical exposure scenarios represent what can happen under routine operations, the probability that people would receive the doses projected here is quite low. There are no residents, hikers, or berrypickers in the vicinity of the majority of treatment units. Precautions such as posting the area are normally used to ensure that no one would be exposed during or immediately after an herbicide application operation.

As described in section 4, these typical scenarios use a number of conservative assumptions that tend to overestimate rather than underestimate what is expected in the majority of operations. For example, predicted levels in water (which determine doses for drinking water and eating fish) are much higher than levels seen in extensive field testing. Extensive monitoring studies conducted by the Forest Service in the Pacific

Table 5-6

Lowest margins of safety for workers for typical and maximum exposures in routine operations

Chemical	Typical Exposures		Maximum Exposures	
	Systemic	Reproductive	Systemic	Reproductive
2,4-D (amine)	16	81	2.2	11
2,4-D (ester)	16	81	1.7	8.4
2,4-DP	9,700	10,000	670	842
Dicamba	1,338 (MML)	254 (MML)	188 (MMAL)	36 (MAML)
Diesel oil	100 (BS)	10,000	21 (MAML)	2,137 (MAML)
Fosamine	120	243	13	25
Glyphosate	600	194	31	10
Hexazinone	500 (MML)	2,521 (MML)	8.3 (MAML)	42 (MAML)
Imazapyr	9,700	5,832	1,000	607
Kerosene	380	10,000	9.8 (MAML)	264 (MAML)
Limonene	4,900	4,903	250 (MAML)	251 (MAML)
Picloram	10,000	10,000	3,100	10,000
Sulfometuron methyl	810	8,100	40	404
Tebuthiuron	61	24	7.2	2.9
Triclopyr (amine)	180 (MAML & MML)	179 (MML)	9.4 (MAML)	9.4 (MAML)
Triclopyr (ester)	290	295	7.5 (MAML)	7.5 (MAML)

Note: Lowest MOS's are for backpack sprayers except as indicated in parentheses--(BS) Basal Stem Applicators, (MAML) Mechanical Applicator-Mixer/Loader, (MML) Mechanical Mixer/Loader.

Table 5-7

Worker spill accident MOS's and LD$_{50}$ (mg/kg)/dose comparisons

Chemical	Systemic MOS	Reproductive MOS	LD$_{50}$ (mg/kg)	Dose (mg/kg)	Ratio of Dose of LD$_{50}$
2,4-D (amine)	-140	-27	375	136.8	0.365
2,4-D (ester)	-140	-27	375	136.8	0.365
2,4-DP	2.3	2.8	532	2.22	0.004
Dicamba	-2.6	-13.9	757	41.76	0.055
Diesel oil	-140	-1.4	7,380	1,020	0.138
Fosamine	-9.6	-4.8	24,400	240	0.010
Glyphosate	-5.8	-18	4,320	180	0.042
Hexazinone	-12	-2.4	1,690	120	0.071
Imazapyr	4.2	2.5	>5,000	120	<0.024
Kerosene	-12	2.2	>28,000	340.5	<0.012
Limonene	-1.9	-1.9	5,000	432	0.086
Picloram	24	172	8,200	0.29	0.000035
Sulfometuron methyl	-84	-8.4	>5,000	210	<0.042
Tebuthiuron	2.5	-1.02	644	5.1	.008
Triclopyr (amine)	-12	-12	630	29.7	0.047
Triclopyr (ester)	-16	-16	630	39.6	0.063

NA = not applicable.

Table 5-8

2,4-D amine margins of safety

Exposure Type	Systemic		Reproductive	
	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	8500.0	3600.0	10000.0	10000.0
Onsite	1100.0	700.0	5570.4	3481.5
Dietary				
Water	10000.0	7000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	180.0	10000.0	880.7
Vegetable	2100.0	940.0	10000.0	4683.1
Berry picking	2500.0	21.0	10000.0	107.3
Workers				
Aerial				
Pilot	160.0	21.0	790.3	103.5
Mixer/Loader	64.0	14.0	317.5	72.3
Observer	4900.0	640.0	10000.0	3188.8
Mechanical ground				
Applicator	190.0	15.0	934.8	72.9
Mixer/loader	79.0	14.0	392.8	71.6
Appl-mix/load	80.0	10.0	398.7	51.9
Manual ground				
Backpack	16.0	2.2	81.0	11.2
Cut surface	40.0	13.0	198.2	63.0
Accidents				
Spill onto worker		-140.0		-27.4
Accidental spray		10.0		49.9
Spills into water				
Ground--18.9 l into pond		29.0		142.9
Air--379 l into reservoir		430.0	2172.4	

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 1 and a reproductive NOEL of 5.

Table 5-9

2,4-D ester margins of safety

Exposure Type	Systemic Typical	Systemic Maximum	Reproductive Typical	Reproductive Maximum
Public				
Dermal				
Drift	5300.0	2100.0	10000.0	10000.0
Onsite	700.0	400.0	3481.5	1989.4
Dietary				
Water	9900.0	4000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	100.0	10000.0	503.2
Vegetable	1300.0	540.0	6475.1	2676.0
Berry picking	1600.0	12.0	7820.2	61.3
Workers				
Aerial				
Pilot	130.0	14.0	632.2	69.0
Mixer/Loader	51.0	9.6	254.0	48.2
Observer	3900.0	430.0	10000.0	2125.9
Mechanical ground				
Applicator	160.0	10.0	779.0	52.1
Mixer/loader	65.0	10.0	327.3	51.1
Appl-mix/load	66.0	7.4	332.2	37.1
Manual ground				
Backpack	16.0	1.7	81.0	8.4
Basal stem	100.0	43.0	508.2	215.5
Accidents				
Spill onto worker		-140.0		-27.4
Accidental spray		6.7		33.3
Spills into water				
Ground--18.9 l into pond		29.0		142.9
Air--379 l into reservoir		290.0		1448.3

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 1 and a reproductive NOEL of 5.

Table 5-10

2,4-DP margins of safety

	Systemic		Reproductive	
Exposure Type	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	10000.0	10000.0	10000.0	10000.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	700.0	10000.0	872.2
Vegetable	6500.0	3100.0	8093.9	3902.6
Berry picking	7800.0	72.0	9775.3	89.4
Workers				
Aerial				
Pilot	10000.0	7900.0	10000.0	9933.8
Mixer/Loader	10000.0	5600.0	10000.0	6944.4
Observer	10000.0	10000.0	10000.0	10000.0
Mechanical ground				
Applicator	10000.0	3600.0	10000.0	4557.3
Mixer/loader	10000.0	3600.0	10000.0	4474.1
Appl-mix/load	10000.0	2600.0	10000.0	3242.2
Manual ground				
Backpack	9700.0	670.0	10000.0	842.4
Basal stem	10000.0	8000.0	10000.0	9958.8
Accidents				
Spill onto worker		2.3		2.8
Accidental spray		2381.0		2976.0
Spills into water				
Ground--18.9 l into pond		150.0		183.5
Air--379 l into reservoir		1700.0		2172.4

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 5 and a reproductive NOEL of 6.25.

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Table 5-11

Dicamba margins of safety

Exposure Type	Systemic Typical	Systemic Maximum	Reproductive Typical	Reproductive Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	10000.0	10000.0	3614.5	2400.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	3615.6	10000.0	686.5
Vegetable	10000.0	10000.0	7692.3	3750.0
Berry picking	10000.0	452.2	9375.0	85.9
Workers				
Mechanical ground				
Applicator	3185.5	264.8	604.8	50.3
Mixer/loader	1337.8	260.0	254.0	49.4
Appl-mix/load	1357.4	188.4	257.7	35.8
Manual ground				
Cut surface	2051.9	522.0	389.6	99.1
Accidents				
Spill onto worker		-2.6		-13.9
Accidental spray		181.4		34.4
Spills into water				
Ground--18.9 l into pond		1717.4		326.1
Air--379 l into reservoir		NA		NA

NA = not applicable.

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 15.8 and a reproductive NOEL of 3.0.

Table 5-12

Diesel margins of safety

Exposure Type	Systemic		Reproductive	
	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	7300.0	10000.0	10000.0
Onsite	2500.0	1400.0	10000.0	10000.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	980.0	10000.0	10000.0
Vegetable	10000.0	7900.0	10000.0	10000.0
Berry picking	10000.0	180.0	10000.0	10000.0
Workers				
Aerial				
Pilot	1900.0	160.0	10000.0	10000.0
Mixer/loader	750.0	110.0	10000.0	10000.0
Observer	10000.0	4800.0	10000.0	10000.0
Mechanical ground				
Applicator	280.0	30.0	10000.0	3004.0
Mixer/loader	120.0	29.0	10000.0	2949.2
Appl-mix/load	120.0	21.0	10000.0	2137.1
Manual ground				
Basal stem	100.0	32.0	10000.0	3255.6
Accidents				
Spill onto worker		-140.0		-1.4
Accidental spray		47.0		4801.5
Spills into water				
Ground--18.9 l into pond		120.0		10000.0
Air--379 l into reservoir		8600.0		10000.0

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 7.38 and a reproductive NOEL of 751.

Table 5-13

Fosamine margins of safety

Exposure Type	Systemic Typical	 Maximum	Reproductive Typical	 Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	5400.0	3500.0	10000.0	6963.8
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	1300.0	10000.0	2651.1
Vegetable	10000.0	7800.0	10000.0	10000.0
Berry picking	10000.0	180.0	10000.0	357.7
Workers				
Aerial				
Pilot	790.0	120.0	1580.8	248.3
Mixer/Loader	320.0	87.0	635.1	173.6
Observer	10000.0	3800.0	10000.0	7657.0
Mechanical ground				
Applicator	3600.0	73.0	7235.9	145.8
Mixer/loader	1500.0	72.0	3041.4	143.2
Appl-mix/load	1500.0	52.0	3086.4	103.7
Manual ground				
Backpack	120.0	13.0	243.0	25.3
Accidents				
Spill onto worker		-9.6		-4.8
Accidental spray		60.0		119.9
Spills into water				
Ground--18.9 l into pond		680.0		1358.7
Air--379 l into reservoir		4300.0		8620.7

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 25 and a reproductive NOEL of 50.

Table 5-14

Glyphosate margins of safety

Exposure Type	Systemic		Reproductive	
	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	10000.0	10000.0	10000.0	4177.8
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	4900.0	10000.0	1590.3
Vegetable	10000.0	10000.0	10000.0	9366.2
Berry picking	10000.0	670.0	10000.0	214.6
Workers				
Aerial				
Pilot	10000.0	380.0	4214.8	124.2
Mixer/loader	5200.0	270.0	1693.5	86.8
Observer	10000.0	10000.0	10000.0	3826.5
Mechanical ground				
Applicator	7700.0	68.0	2492.9	21.9
Mixer/loader	3200.0	67.0	1047.4	21.5
Appl-mix/load	3300.0	48.0	1063.1	15.6
Manual ground				
Backpack	600.0	31.0	194.4	10.1
Cut surface	930.0	300.0	301.3	95.8
Accidents				
Spill onto worker		-5.8		-18.0
Accidental spray		190.0		59.9
Spills into water				
Ground--18.9 l into pond		1100.0		362.1
Air--379 l into reservoir		10000.0		4344.8

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 31 and a reproductive NOEL of 10.

Table 5-15

Hexazinone margins of safety

	Systemic		Reproductive	
Exposure Type	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	9800.0	2800.0	10000.0	10000.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	1100.0	10000.0	5301.0
Vegetable	10000.0	6200.0	10000.0	10000.0
Berry picking	10000.0	140.0	10000.0	715.4
Workers				
Aerial				
Pilot	1300.0	170.0	6322.2	827.8
Mixer/Loader	510.0	120.0	2540.3	578.7
Observer	10000.0	5100.0	10000.0	10000.0
Mechanical ground				
Applicator	1200.0	12.0	5998.9	58.3
Mixer/loader	500.0	11.0	2520.6	57.3
Appl-mix/load	510.0	8.3	2558.2	41.5
Manual ground				
Backpack	970.0	10.0	4860.2	50.5
Soil spot	900.0	290.0	4519.0	1437.1
Accidents				
Spill onto worker		-12.0		-2.4
Accidental spray		80.0		399.6
Spills into water				
Ground--18.9 l into pond		540.0		2715.5
Air--379 l into reservoir		5800.0		10000.0

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 10 and a reproductive NOEL of 50.

Table 5-16

Imazapyr margins of safety

	Systemic		Reproductive	
Exposure Type	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	10000.0	10000.0	10000.0	10000.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	10000.0	10000.0	10000.0
Vegetable	10000.0	10000.0	10000.0	10000.0
Berry picking	10000.0	10000.0	10000.0	10000.0
Workers				
Aerial				
Pilot	10000.0	10000.0	10000.0	9933.8
Mixer/loader	10000.0	10000.0	10000.0	6944.4
Observer	10000.0	10000.0	10000.0	10000.0
Mechanical ground				
Applicator	10000.0	2300.0	10000.0	1400.0
Mixer/loader	10000.0	2300.0	10000.0	1374.4
Appl-mix/load	10000.0	1700.0	10000.0	996.0
Manual ground				
Backpack	9700.0	1000.0	5832.3	606.6
Cut surface	10000.0	8200.0	10000.0	4927.1
Accidents				
Spill onto worker		4.2		2.5
Accidental spray		8000.0		4795.2
Spills into water				
Ground--18.9 l into pond		10000.0		10000.0
Air--379 l into reservoir		10000.0		10000.0

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 500 and a reproductive NOEL of 300.

Table 5-17

Kerosene margins of safety

Exposure Type	Systemic Typical	Systemic Maximum	Reproductive Typical	Reproductive Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	8200.0	4100.0	10000.0	10000.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	2900.0	10000.0	10000.0
Vegetable	10000.0	10000.0	10000.0	10000.0
Berry picking	10000.0	530.0	10000.0	10000.0
Workers				
Aerial				
Pilot	1900.0	120.0	10000.0	3286.5
Mixer/loader	750.0	86.0	10000.0	2297.5
Observer	10000.0	3800.0	10000.0	10000.0
Mechanical ground				
Applicator	1100.0	14.0	10000.0	370.5
Mixer/loader	470.0	14.0	10000.0	363.8
Appl-mix/load	470.0	9.8	10000.0	263.6
Manual ground				
Backpack	380.0	27.0	10000.0	714.5
Basal stem	1100.0	360.0	10000.0	9752.4
Accidents				
Spill onto worker		-12.0		2.2
Accidental spray		59.0		1586.4
Spills into water				
Ground--18.9 l into pond		1300.0		10000.0
Air--379 l into reservoir		10000.0		10000.0

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 28 and a reproductive NOEL of 751.

Table 5-18

Limonene margins of safety

Exposure Type	Systemic		Reproductive	
	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	10000.0	10000.0	10000.0	10000.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	10000.0	10000.0	10000.0
Vegetable	10000.0	10000.0	10000.0	10000.0
Berry picking	10000.0	5400.0	10000.0	5413.4
Workers				
Aerial				
Pilot	10000.0	3100.0	10000.0	3131.9
Mixer/loader	10000.0	2200.0	10000.0	2189.4
Observer	10000.0	10000.0	10000.0	10000.0
Mechanical ground				
Applicator	10000.0	350.0	10000.0	353.1
Mixer/loader	10000.0	350.0	10000.0	346.7
Appl-mix/load	10000.0	250.0	10000.0	251.2
Manual ground				
Backpack	4900.0	510.0	4903.4	510.0
Basal stem	7300.0	2300.0	7306.4	2323.4
Accidents				
Spill onto worker		-1.9		-1.9
Accidental spray		1500.0		1511.8
Spills into water				
Ground--18.9 l into pond		3400.0		3424.6
Air--379 l into reservoir		10000.0		10000.0

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 227 and a reproductive NOEL of 227.

Table 5-19

Picloram margins of safety

Exposure Type	Systemic Typical	Systemic Maximum	Reproductive Typical	Reproductive Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	10000.0	10000.0	10000.0	10000.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	4200.0	10000.0	10000.0
Vegetable	10000.0	10000.0	10000.0	10000.0
Berry picking	10000.0	430.0	10000.0	3066.1
Workers				
Aerial				
Pilot	10000.0	10000.0	10000.0	10000.0
Mixer/Loader	10000.0	9000.0	10000.0	10000.0
Observer	10000.0	10000.0	10000.0	10000.0
Mechanical ground				
Applicator	10000.0	9700.0	10000.0	10000.0
Mixer/loader	10000.0	9500.0	10000.0	10000.0
Appl-mix/load	10000.0	6900.0	10000.0	10000.0
Manual ground				
Backpack	10000.0	3100.0	10000.0	10000.0
Cut surface	10000.0	10000.0	10000.0	10000.0
Accidents				
Spill onto worker		24.0		171.5
Accidental spray		6200.0		10000.0
Spills into water				
Ground--18.9 l into pond		2800.0		10000.0
Air--379 l into reservoir		8100.0		10000.0

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 7 and a reproductive NOEL of 50.

Table 5-20

Sulfometuron methyl margins of safety

Exposure Type	Systemic		Reproductive	
	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	10000.0	10000.0	10000.0	10000.0
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	4300.0	10000.0	10000.0
Vegetable	10000.0	10000.0	10000.0	10000.0
Berry picking	10000.0	580.0	10000.0	5800.8
Workers				
Aerial				
Pilot	3600.0	310.0	10000.0	3104.3
Mixer/Loader	1500.0	220.0	10000.0	2170.1
Observer	10000.0	9600.0	10000.0	10000.0
Mechanical ground				
Applicator	5000.0	300.0	10000.0	2956.1
Mixer/loader	2100.0	290.0	10000.0	2902.1
Appl-mix/load	2100.0	210.0	10000.0	2103.0
Manual ground				
Backpack	810.0	40.0	8100.4	404.4
Accidents				
Spill onto worker		-84.0		-8.4
Accidental spray		150.0		1498.5
Spills into water				
Ground--18.9 l into pond		78.0		775.9
Air--379 l into reservoir		10000.0		10000.0

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 2.5 and a reproductive NOEL of 25.

Table 5-21

Tebuthiuron margins of safety

	Systemic		Reproductive	
Exposure Type	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	10000.0	3500.0	8333.3	1392.8
Dietary				
Water	10000.0	10000.0	10000.0	10000.0
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	1300.0	10000.0	530.2
Vegetable	10000.0	10000.0	10000.0	9615.4
Berry picking	10000.0	180.0	10000.0	71.5
Workers				
Aerial				
Pilot	2000.0	100.0	789.9	41.4
Mixer/loader	790.0	72.0	317.5	28.9
Observer	10000.0	3200.0	10000.0	1275.5
Mechanical ground				
Applicator	3500.0	73.0	1400.6	29.2
Mixer/loader	1500.0	72.0	588.9	28.6
Appl-mix/load	1500.0	52.0	598.1	20.8
Manual ground				
Backpack	61.0	7.2	24.3	2.9
Soil Spot	10000.0	8500.0	10000.0	3378.4
Accidents				
Spill onto worker		2.5		-1.02
Accidental spray		50.0		20.0
Spills into water				
Ground--18.9 l into pond		160.0		63.9
Air--379 l into reservoir		3600.0		1428.6

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 12.5 and a reproductive NOEL of 50.

Table 5-22

Triclopyr amine margins of safety

Exposure Type	Systemic		Reproductive	
	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	6300.0	3200.0	6330.0	3165.0
Dietary				
Water	10000.0	8700.0	10000.0	8706.2
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	250.0	10000.0	249.3
Vegetable	3200.0	1200.0	3237.5	1170.8
Berry picking	3900.0	27.0	3910.1	26.8
Workers				
Aerial				
Pilot	960.0	130.0	957.9	125.4
Mixer/Loader	380.0	88.0	384.9	87.7
Observer	10000.0	3900.0	10000.0	3865.2
Mechanical ground				
Applicator	420.0	13.0	424.9	13.3
Mixer/loader	180.0	13.0	178.5	13.0
Appl-mix/load	180.0	9.4	181.2	9.4
Manual ground				
Backpack	210.0	31.0	210.4	30.6
Cut surface	550.0	130.0	547.8	129.0
Accidents				
Spill onto worker		-12.0		-11.9
Accidental spray		61.0		60.5
Spills into water				
Ground--18.9 l into pond		91.0		90.5
Air--379 l into reservoir		720.0		724.1

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 2.5 and a reproductive NOEL of 2.5.

Table 5-23

Triclopyr ester margins of safety

Exposure Type	Systemic		Reproductive	
	Typical	Maximum	Typical	Maximum
Public				
Dermal				
Drift	10000.0	10000.0	10000.0	10000.0
Onsite	6300.0	3200.0	6330.0	3165.0
Dietary				
Water	10000.0	8700.0	10000.0	8706.2
Fish	10000.0	10000.0	10000.0	10000.0
Meat	10000.0	250.0	10000.0	249.3
Vegetable	3200.0	1200.0	3237.5	1170.8
Berry picking	3900.0	27.0	3910.1	26.8
Workers				
Aerial				
Pilot	1400.0	94.0	1436.9	94.1
Mixer/loader	580.0	66.0	577.3	65.8
Observer	10000.0	2900.0	10000.0	2898.9
Mechanical ground				
Applicator	850.0	11.0	849.8	10.6
Mixer/loader	360.0	10.0	357.1	10.4
Appl-mix/load	360.0	7.5	362.4	7.5
Manual ground				
Backpack	290.0	20.0	294.6	20.4
Basal stem	880.0	280.0	877.8	279.1
Accidents				
Spill onto worker		-16.0		-15.8
Accidental spray		45.0		45.4
Spills into water				
Ground--18.9 l into pond		68.0		67.9
Air--379 l into reservoir		540.0		543.1

Note: Margins of safety greater than 10,000 are listed as 10000. Margins of safety were based on a systemic NOEL of 2.5 and a reproductive NOEL of 2.5.

Northwest for phenoxy herbicides from 1974 to 1978 showed negligible levels of herbicides in streams (all were less than 0.04 ppm). These extremely low levels were found despite the fact that during the 1974-78 period not all herbicide applications were monitored. Only those applications most likely to result in significant residues or cause for public concern were actually monitored (USDA, 1980).

The levels predicted on berries also are higher than those found in similar forest plants (USDA, 1984). In addition, the levels predicted for deer meat in the typical exposures are similar to the highest levels found by Newton and Norris (1968, as cited in Dost, 1983), who found no levels greater than 0.08 ppm in edible deer tissues.

Risk to the Public From Maximum Exposures

The maximum exposure scenarios described in section 4 were intended to indicate the upper bound for public exposure to herbicide applications in the Southeast. The low probability of each assumption, which would apply to all of the events that led to the MOS's described in table 5-2, must be emphasized. It is unlikely that anyone would receive a dose as high as those estimated here.

Margins of Safety for Maximum Public Exposures. Table 5-2 indicates that most margins of safety for systemic and reproductive effects estimated for maximum public exposures are greater than 100 except for 2,4-D amine and ester, 2,4-DP, dicamba, tebuthiuron, and triclopyr amine and ester. Margins of safety for systemic effects calculated for exposure to 2,4-D were 12 for the ester and 21 for the amine. MOS's were 27 for both triclopyr amine and ester. The 2,4-DP MOS for systemic effects (72) indicates some risk, though not nearly as great as the risks from 2,4-D and triclopyr. Chronic doses of 2,4-D, as predicted by this analysis, could affect the muscles and kidneys, though this would be a reversible effect. People who chronically receive the maximum triclopyr doses predicted here could experience decreased kidney function. Because the margins of safety were computed by comparing acute exposures with chronic no-effect levels, the risk of occurrence of these effects should be considered extremely low. It is extremely unlikely that nearby residents would receive repeated maximum doses over the long term. The margins of safety derived for triclopyr amine and ester also are conservative because the toxic effects observed in dogs that resulted in a systemic NOEL of 2.5 mg/kg/day may have been exacerbated by the decreased renal excretion capacity of dogs, which is not representative of human renal physiology. A 2-year feeding study in rats did not result in kidney problems or other toxic effects at a comparable dose level (3 mg/kg/day) (USDA, 1984).

Lowest MOS's for reproductive effects are 27 for both triclopyr amine and ester, indicating some risk of reproductive effects under maximum exposure situations. The reproductive MOS's for 2,4-D ester (61), tebuthiuron (72) dicamba (86), and 2,4-DP (89) indicate somewhat lower risks. Reproductive effects risks for the general public for the other 10 chemicals are low to negligible because MOS's range from 140 to more than 10,000. Thus, these 10 chemicals can be considered safe for the public even if exposed to the relatively high amounts predicted under the maximum exposure assumptions and even if the maximum exposures occur repeatedly.

Probability of the Maximum Public Exposures Occurring. The probability of someone receiving a dose as high as those predicted in the maximum exposure estimate is negligible. The probability is so low because the analysis assumes that a number of unlikely events occur simultaneously. For example, using the assumptions that for a project on a given day the probability of treating a unit with the maximum acreage at the maximum application rate is 1 in 100, and the probability of the high drift case is 1 in 100, and the probability of someone being in the vicinity of the treatment area is 1 in 100, then the probability of someone receiving a dose as high as those predicted here is 1 in 1 million (0.01 x 0.01 x 0.01 = 0.000001).

Risk to the Public From Accidents

Table 5-3 summarizes the risk to the public from accidents: direct exposure to aerial applications or drinking water from a pond or reservoir that has received an herbicide spill. The low margin of safety for 2,4-D (6.7) indicates that people exposed to spray from a direct aerial application could experience systemic or reproductive toxic effects. The risks of 2,4-D reproductive effects would be somewhat lower. The severity of effects would depend upon what measures were taken after the spraying incident. The public would not likely be affected if they wash immediately. The longer washing is postponed, the more serious the effects are likely to be. Triclopyr ester (45), triclopyr amine (61), diesel oil (MOS = 47), fosamine (MOS = 60), hexazinone (MOS = 80), kerosene (MOS = 59), and tebuthiuron (MOS = 50) also present a risk of systemic effects from direct spraying, though not as great a risk as those described for 2,4-D. Dicamba, glyphosate, tebuthiuron, and triclopyr present a risk of reproductive effects in this situation.

Spills into reservoirs present negligible risk to the public. The lowest systemic margin of safety is 290 for 2,4-D. Pond spills of 2,4-D (MOS = 29) do present a risk of systemic effects, such as those described in table 5-5, to the public. Pond spills of triclopyr amine and ester (MOS's = 91 and 68, respectively) and sulfometuron methyl (MOS = 78) are lower systemic risk situations. Triclopyr amine, ester, and tebuthiuron present the only risks of reproductive effects from pond spills. Normal spill cleanup procedures and warning signs should prevent any of these possible effects from occurring. None of the other chemicals present a risk to the general public in a pond spill, although in rare instances sensitive individuals could be at risk.

Again it must be noted that these are one-time, rather than repeat, or chronic, exposures and that comparison of these doses with acute LD_{50}'s shows that no one is likely to be at risk of fatal effects. For example, the lowest MOS (6.7) for the public is for direct spraying with 2,4-D ester. This dose is less than 1/2,100 of the LD_{50}. Complete margins of safety computed for each chemical for accidents are presented in tables 5-8 to 5-23.

Probability of Accidents. The risk of a member of the public being hit directly by an aerial spray operation is very small. The probability of a pesticide application in an area not scheduled for treatment is low. According to the Forest Service data on insecticide application (USDA, 1984), an estimate of the probability, based on empirical data, of some kind of significant error in a pesticide application is 0.0002 (at the 95-percent confidence level). Operational features of herbicide applications make the probability of applying an herbicide in an area not scheduled for treatment less than that of insecticide operations. Using this value as an extremely conservative estimate of the probability of an application directly hitting a human, there might be three accidents over a period of 8 years if a spraying operation occurred every day for 6 months during each of those years. In addition, the probability that someone would be in the area being sprayed is very low because normally the area is posted before spraying and humans are kept out of the treated areas during spray operations. Thus, the probability of such accidents can be considered negligible.

Some indication of the likelihood of occurrence of significant herbicide spill accidents may be derived from historical data. Herbicide spill accidents recorded by BLM and the Forest Service in the Pacific Northwest over 11 years were classified by location, date, and quantity spilled. Also included was information specifying whether the accidents occurred on the ground or in the air, and whether the spill was near a waterway. Over an 11-year period, from 1973 through 1983, there were 24 recorded spills averaging 44.4 gallons per accident. Herbicide use rates ranged from 1.5 pound a.i. to 7 pounds a.i. per acre for normal use rates. For a total of 302,085 acres sprayed during the 11-year period, there was one accident for every 12,587 acres, and 54 percent of the spills involved 30 gallons or less. Table 5-4 shows the acreage sprayed, gallons spilled, and type of spill for the years 1973 to 1983.

Risk to the Public and Workers From Herbicides Used in Brown-and-Burn Operations

The analysis of risk from brown-and-burn operations is not based on the calculation of a margin-of-safety based on laboratory NOEL. It is based on the calculation of a ratio between an estimated safe human inhalation dose and the estimated exposure. Reference levels based on threshold limit values (safety guidelines for occupational exposure), inhalation studies in laboratory animals, or manufacturers' information were used to calculate margins of safety for brown-and-burn herbicide exposures. The threshold limit value (TLV) is the time-weighted average concentration in air of a chemical for a normal 8-hour workday and a 40-hour workweek, to which nearly all workers may be repeatedly exposed, day after day, without adverse effect (ACGIH, 1984). For those herbicides where TLV's or other similar criteria were not available (glyphosate and imazapyr), a safety factor of 1,000 was applied to the rat-inhalation LC_{50} value (1/1,000 the LC_{50}) to estimate a safe exposure level. There are no inhalation studies available for 2,4-DP. Herbicide concentrations in air would dissipate with distance from the burn site, and the public would be expected to have lower exposures than the workers.

An example of the estimated onsite herbicide concentrations in air is given for hexazinone in table 5-24. This table shows the types of exposures that were calculated for the herbicides used in brown-and-burn operations as described in section 4. The concentration in air is for a maximum respirable level, assuming no dissipation or transport from the burn site.

Triclopyr ester has the highest concentration in air (0.13 mg/m^3) of any of the herbicides under typical conditions. Ratios of reference levels to doses are all greater than or equal to 150 for typical conditions, except triclopyr ester, which has a ratio of 34 for aerial foliar and mechanical foliar methods. Under the minimum time interval conditions, triclopyr ester has the highest concentrations of any of the herbicides (see table 5-25). Ratios are all greater than 100, except for triclopyr ester, which result in a value of 31 and triclopyr amine, with a ratio of 71 (see table 5-25).

The estimated doses are undoubtedly higher than those likely to occur because a large fraction of the herbicide residues would probably be destroyed during combustion (McMahon et al., 1985; Bush et al., 1987). McMahon et al. (1985) determined that more than 95 percent decomposition of herbicide residues (including 2,4-D, 2,4-DP, picloram, and hexazinone) occurred when treated wood (chestnut oak) was burned under conditions of rapid combustion. Under smoldering conditions, much higher residues were recovered. Combustion during prescribed burns is generally similar to the rapid combustion conditions.

The ratios of reference levels to doses for the wildfire scenario are greater than 100 for all the herbicides except 2,4-D ester, 2,4-DP, imazapyr, triclopyr amine, and triclopyr ester. Wildfire scenarios with ratios of less than 100 are as follows: 2,4-D ester mechanical foliar ratio = 66; 2,4-DP mechanical foliar ratio = 55; imazapyr aerial foliar, mechanical foliar, foliar B/P hand ratios = 46; triclopyr amine mechanical foliar ratio = 66; and triclopyr ester aerial foliar and mechanical foliar ratios = 66.

The estimated wildfire exposures represent maximum values based on typical application rates and assuming no degradation between treatment and the time of burning. Under smoldering conditions, exposures are not likely to be reduced significantly for stable compounds, such as 2,4-D, but would probably be much less for thermally unstable compounds, such as picloram (Bush et al., 1987). If rapid combustion occurs, residues and exposures would be lower, as discussed in a preceding paragraph.

Risk to the Public From Using Treated Firewood

Bush et al. (1987) measured residues released from burning wood (in wood stoves or fireplaces) from herbicide-injected trees. Residues under rapid combustion were generally much less than under slow combustion. Based on these measurements, Bush et al. estimated indoor air concentrations of herbicides for rapid and slow combustion conditions, respectively, as follows: 0.0000036 mg/m^3 to 0.000088 mg/m^3 for 2,4-D; 0.00012 mg/m^3 to 0.001 mg/m^3 for 2,4-DP; less than 0.0000001 mg/m^3 for picloram; and less than 0.00005 mg/m^3 to 0.00031 mg/m^3 for triclopyr (Bush et al., 1987).

Table 5-24

Brown-and-burn exposures and risk evaluation for hexazinone

Application Method	Concentration in Air (mg/m^3)	Risk Ratio[a] (AEL/Concentration)
Aerial foliar-typical	0.0035	2,900
Aerial foliar-maximum	0.011	910
Mechanical foliar-typical	0.0013	7,800
Mechanical foliar-maximum	0.011	910
Mechanical G/P[b]-typical	0.000062	160,000
Mechanical G/P[b]-maximum	0.011	910
Manual Ground G/P[b]-typical	0.0035	2,900
Manual Ground G/P[b]-maximum	0.011	910
Foliar BP[c]/hand-typical	0.000022	450,000
Foliar BP[c]/hand-maximum	0.00041	24,000
Basal stem-typical	--[d]	--
Basal stem-maximum	--	--
Cut surface-typical	--	--
Cut surface-maximum	--	--

[a] Risk ratios are based on the AEL (acceptable exposure level) of 10.0 mg/m^3 (DuPont, 1987) which is a time-weighted average value. A TLV is not available for hexazinone.

[b] G/P = Granular/pellet.

[c] BP = Backpack.

[d] -- = hexazinone not used in these methods.

Table 5-25

Maximum exposures for each herbicide used in brown-and-burn operations

Herbicide	Application Method	Concentration in Air (mg/m^3)	Reference Level (mg/m^3)	Ratio (Reference Level/ Concentration)
2,4-D Amine	Mechanical foliar-maximum	0.026	10.0[a]	380
2,4-D Ester	Mechanical foliar-maximum	0.042	10.0[a]	240
2,4-DP	Mechanical foliar-maximum	0.042	NA[b]	--
Glyphosate	Aerial foliar-maximum	0.013	>12.2[c]	>950
Hexazinone	Aerial foliar-maximum	0.011	10.0[d]	910
	Mechanical foliar-maximum	0.011	10.0[d]	910
	Mechanical G/P-maximum	0.011	10.0[d]	910
	Manual ground G/P-maximum	0.011	10.0[d]	910
Imazapyr	Aerial foliar-maximum	0.013	>1.3[e]	>100
	Mechanical foliar-maximum	0.013	>1.3[e]	>100
Limonene	All methods	0.034	5.0[f]	150
Picloram	Mechanical foliar-maximum	0.0033	10.0[a]	3,000
Sulfometuron methyl	Foliar BP/hand-maximum	0.000000068	10.0[g]	1,500,000,000
Triclopyr amine	Mechanical foliar-maximum	0.056	4.0[h]	71
Tricopyr ester	Aerial foliar-maximum	0.13	4.0[h]	31
	Mechanical foliar-maximum	0.13	4.0[h]	31

Source:

[a] TLV (personal communication ACGIH, Kim Stewart, July 14, 1987).

[b] No inhalation studies are available for 2,4-DP toxicity.

[c] Based on a rat inhalation LC_{50} of >12,200 mg/m^3 (WSSA, 1983) and a safety factor of 1,000.

[d] AEL - Acceptable Exposure Level, DuPont, 1987.

[e] Based on a rat inhalation LC_{50} of >1,300 mg/m^3 (American Cyanamid, 1985) and a safety factor of 1,000.

[f] Based on a rat inhalation LC_{50} of >5,000 mg/m^3 (EPA, 1984c) and a safety factor of 1,000.

[g] AEL (Dupont, 1986).

[h] AEL (personal communication, Dow, Sue McCollister, November 2, 1988).

These concentrations are much less than the maximum exposure concentrations estimated for these herbicides in brown-and-burn operations (see table 5-24).

Risk to Workers From Routine Operations

Table 5-6 lists the lowest margins of safety for workers for typical and maximum exposures based on the lowest systemic and reproductive NOEL's for the 11 herbicides and 3 related chemicals. Full tables showing margins of safety computed for the 14 herbicides and additives are presented in tables 5-8 through 5-23. Because of the assumptions that were made to overestimate risk, the Forest Service estimates that exposures in almost all of the operations that take place will be less than or equal to the typical exposure estimates. The typical worker exposures and resultant margins of safety are what could be expected in the majority of vegetation management programs in the Southeast for workers wearing protective clothing or equipment.

Effects of the Use of Protective Clothing

The use of protective clothing can substantially reduce worker doses, as shown in field studies of worker exposure, and thereby increase their margins of safety. Protective clothing can reduce worker exposures by 27 to 99 percent, as shown in a number of relevant field studies. Typical exposures were computed assuming protective clothing is worn. The calculated maximum doses were based on the assumption that workers work with bare hands and wear ordinary work clothing, such as cotton pants and short-sleeve shirts. The Forest Service requires employees applying herbicides to wear clothing that affords more protection. Typical protective clothing often includes long-sleeve shirts or coveralls, gloves, and hats.

Research has shown that such protective clothing can substantially reduce worker exposure. For example, in right-of-way spraying, doses received by spray gun applicators wearing clean coveralls and gloves were reduced by 68 percent compared to doses without this protection (Libich et al., 1984) During an aerial spraying operation, mixer/loaders wearing protective clothing reduced their exposure by 27 percent and other crew members reduced their exposure by 58 percent compared to the levels observed without precautions (Lavy et al., 1982).

During insecticide applications to orchards, mixers reduced their exposure by 35 percent and sprayers reduced their exposure by 49 percent by wearing coveralls (Davies et al., 1982). Putnam and coworkers found that nitrofen applicators and mixer/loaders wearing protective clothing reduced their exposure by 94 to 99 percent compared to the doses experienced without protection (Waldron, 1985). Although protective clothing generally reduces worker exposure and resulting doses, the degree of protection depends on the application system, the work practices, and the specific herbicide. In one extreme case, workers wearing protective clothing did not receive significantly lower doses than workers with less clothing (Lavy et al., 1984). In this case, backpack applicators had to treat and move through dense vegetation that was taller than themselves.

Most exposure to herbicide applicators is dermal, not inhalation (Kolmodin-Hedman, et al., 1983), so the use of respirators is often ineffective and unnecessary. The hands are the site of the greatest potential herbicide exposure, and rubber gloves are generally quite effective in preventing exposure to hands (Putnam et al., 1983).

Based on the review of field studies, protective clothing was normally found to reduce worker doses by the following amounts:

Type of Worker	Percent Reduction in Dose
1. Mechanical-ground	68.1
2. Aerial application crew members	57.1
3. Aerial mixer/loaders	27.1
4. Injection bar applicators	54.7
5. Hack-and-squirt applicators	57.6

Risk to Workers From Typical Exposures

For typical exposures, all categories of workers applying 2,4-DP, diesel oil, fosamine, glyphosate, hexazinone, imazapyr, kerosene, limonene, picloram, sulfometuron methyl, and triclopyr have MOS's greater than 100. This indicates that even workers chronically exposed to these chemicals should suffer no ill effects. For workers applying 2,4-D and tebuthiuron, at least one category of worker (primarily backpack sprayers) had MOS's less than 100. This means that unprotected workers who routinely receive doses this high may experience some toxic effects from applying these herbicides.

Backpack sprayers are clearly at greatest risk based on comparisons of estimated doses with NOEL's for all of the herbicides. Cut-surface applicators are next, while mixer/loaders and applicator-mixer/loaders for mechanical applications are at somewhat lower risk. Aerial application personnel are at least risk.

Risk to Workers From Maximum Exposures

As shown in table 5-6, a number of herbicides have margins of safety less than 10 for the maximum worker exposures.

Backpack sprayers using 2,4-D and tebuthiuron are at highest risk. However, none of the maximum doses exceeds the lowest NOEL.

The maximum exposures for workers are based on a series of assumptions that, acting together, greatly increase the estimated risk. The analysis uses the highest application rates used by the Forest Service, and the longest work hours for each type of project.

The probability of workers receiving repeated daily doses as high as predicted here is extremely low (less than 1 chance in 1,000). These exposures are not likely to occur chronically. Most of the time workers will be receiving doses less than the maximum exposures predicted. Thus, the average worker would not be expected to experience toxic effects (for example, decreased renal function) that have only been observed after chronic exposure. However, other effects (for example, skin irritation, neural or reproductive effects) might possibly occur after short-term exposure to unusually high levels. Sensitive individuals would be at greatest risk of such effects.

Risk to Workers From Spills of Concentrate on Their Skin

It is important to note that the doses estimated here for workers who spill concentrate on their skin are based on dermal penetration levels derived in studies over many days: the chemicals do not penetrate the skin immediately but over a considerable period of time. Thus, workers would have to ignore their own safety and not wash the chemical off to receive doses as high as predicted in this scenario. All Region 8 application operations have wash water available onsite, and all workers are trained in safety procedures.

For workers who spill 500 ml of concentrate on their skin, there is a clear possibility that they could experience some acute toxic effects if they did not wash it off. The margins of safety for this accidental case are presented in table 5-7. Many of the spill doses approach the LD_{50}. This represents a clear risk of severe toxic effects if the chemical is not washed off. There is some possibility that the damage caused by such a large acute dose could cause long-term damage to vital organs. There have also been rare instances in which limited exposure to 2,4-D was reported (but not conclusively demonstrated) to have caused permanent nerve damage. But, again, it is highly unlikely that a worker would allow the concentrated chemical to penetrate the skin for any length of time.

CANCER RISK

An analysis of the maximum cancer risk was conducted for 2,4-DP since it had positive laboratory oncogenic studies; for the light fuel oils, because they contain small amounts of materials known or suspected of causing cancer; and for the herbicides 2,4-D, glyphosate, and picloram for which there is scientific uncertainty about their ability to cause cancer. There is no evidence to suggest that any of the other chemicals could cause cancer. However, the herbicide fosamine and the adjuvant limonene have not been tested in chronic feeding/oncogenicity studies, and only preliminary oncogenicity study data were available for imazapyr. All of the other herbicides have negative cancer studies. EPA has requested additional data on the cancer potential of a number of the herbicides, and the Forest Service will consider the results of their findings when they become available.

Cancer is generally dealt with in the scientific community as a nonthreshold response, which means that even an extremely small amount of a chemical could cause a tumor. The multistage model used for estimating the risk for all herbicides in this analysis is a reasonably conservative estimator used by EPA. At high doses, all of the commonly used models would predict nearly the same rate of tumor formation.

Cancer risks for 2,4-DP, 2,4-D, glyphosate, picloram, and the light fuel oils have been calculated based on a variety of conservative assumptions that are likely to overestimate the risks. These assumptions include the following:

1. 2,4-DP, glyphosate, picloram, and 2,4-D are all treated as if they are carcinogenic. Picloram, glyphosate, and 2,4-D have not been shown conclusively to be carcinogenic in laboratory tests, but the evidence did not rule out the possibility of a low carcinogenic potency. Consequently, a conservative approach was taken.

2. In cases where there is more than one data set available, the data set indicating greater carcinogenic potency has been chosen. For example, the carcinogenic potency of 2,4-D and has been calculated based on the rate of tumor formation in the female Osborne-Mendel rats studied by Hansen et al. (1971). This is the species and sex that have exhibited the highest rate of tumor formation after 2,4-D administration. All tumors were considered, although many of them were benign.

3. It is assumed that carcinogenicity in all five cases is not a threshold phenomenon; that is, any dose of these chemicals has some probability of causing cancer, no matter how small the dose.

4. In each case a 95-percent upper confidence limit on the multistage model estimate was used to estimate cancer potency using the maximum-likelihood procedure of the GLOBAL 82 computer program (Howe and Crump, 1982).

5. Interspecies extrapolation is a principal source of uncertainty in judging cancer risk. The scaling method used in this analysis is the most conservative of the commonly accepted methods. The cancer potency of each chemical for humans was assumed to be the same as the potency for rats when scaled in terms of milligrams per square meter (mg/m^2) of body surface area. This method is commonly used by EPA and others, but it is not the only acceptable approach. Another equally acceptable (OSTP, 1985) method is to scale doses in terms of mg/kg of body weight, resulting in estimates of cancer risk that are about 16 percent of those calculated here.

6. The range of doses calculated for workers and the public in the basic scenarios covers even extreme exposures that might be encountered with each application method. Unusual exposure situations, represented by accidental spraying and large herbicide spills, have also been considered.

The probability of occurrence of cancer over a lifetime as a result of exposure to each of the chemicals was calculated using the following equations:

$$P(d) = K \times b \times d$$
$$d = D \times N/L$$

where:

P(d) is a conservative estimate of the probability of cancer during a person's lifetime as the result of dose d.

d is the average daily dose over a lifetime (mg/kg/day)

K is an interspecies extrapolation factor

b is a 95-percent upper confidence limit on the estimate for cancer potency in the test animal (derived in section 3).

The following cancer potencies (per mg/kg/day) were used: 2,4-DP, 0.0124; 2,4-D, 0.00503; picloram, 0.00057; glyphosate, 0.00002566; and kerosene and diesel oil, 0.0000009. These potencies (b) refer to the test animal; the potency for humans is K x b.

D is the daily dose (mg/kg/day)

N is the number of days during which the dose D occurs during an individual's lifetime

L is the number of days in a lifetime, taken to be 25,550 for a 70-year lifespan.

The interspecies extrapolation factor, K, can be estimated by assuming that body surface area is proportional to body weight to the 2/3 power (Mantel and Schneiderman, 1975), so that K would be:

$$K = (\text{human weight/test animal weight})^{1/3}$$

For an average human weight of 50 kilograms and an average rat weight of 350 grams, K is estimated to be 5.2.

Cancer Risk to the Public

Cancer risk for the general public was calculated for a combination of nine typical exposures and one maximum exposure in a lifetime. The approximate upper bound cancer risks to the public for the combined typical and maximum exposures are shown in table 5-26. (See section 4 for details of lifetime exposures of the public.) Public cancer risks are never greater than 2 in 10 million for any of the seven chemicals examined for the nine typical and one maximum lifetime exposures.

Cancer Risk to Workers

Cancer risk to workers has been calculated assuming that typical exposures and days of application per year are experienced during 90 percent of the years, and that during 10 percent of the years, maximum exposures and days of application are experienced. A total of 20 years of employment in herbicide application has been assumed for each worker. The upper bounds for lifetime cancer risks for workers are shown in table 5-26. The risks for each herbicide were calculated assuming that only that herbicide was

Table 5-26

Lifetime cancer risk

	2,4-D Amine	2,4-D Ester	2,4-DP	Diesel	Glyphosate	Kerosene	Picloram
Public							
Dermal							
Drift	1.4×10^{-9}	2.2×10^{-9}	8.9×10^{-11}	8.7×10^{-13}	1.9×10^{-11}	1.0×10^{-12}	1.4×10^{-12}
Onsite	9.8×10^{-9}	1.6×10^{-8}	6.4×10^{-10}	6.2×10^{-12}	1.3×10^{-10}	7.2×10^{-12}	9.6×10^{-12}
Dietary							
Water	7.3×10^{-10}	1.2×10^{-9}	2.9×10^{-9}	1.1×10^{-13}	6.1×10^{-12}	1.3×10^{-13}	2.4×10^{-11}
Fish	1.5×10^{-10}	2.4×10^{-10}	5.7×10^{-10}	1.1×10^{-13}	1.2×10^{-12}	1.3×10^{-13}	4.9×10^{-12}
Meat	6.2×10^{-9}	1.1×10^{-8}	2.0×10^{-8}	1.5×10^{-12}	8.2×10^{-11}	1.9×10^{-12}	2.1×10^{-10}
Vegetable	5.5×10^{-9}	9.0×10^{-9}	2.2×10^{-8}	8.5×10^{-13}	4.6×10^{-11}	9.9×10^{-13}	1.8×10^{-10}
Berry picking	5.1×10^{-8}	9.0×10^{-8}	1.9×10^{-7}	8.4×10^{-12}	6.2×10^{-10}	1.1×10^{-11}	2.0×10^{-9}
Workers							
Aerial							
Pilot	2.0×10^{-6}	9.4×10^{-7}	3.3×10^{-8}	1.0×10^{-10}	9.8×10^{-9}	3.9×10^{-10}	7.2×10^{-10}
Mixer/loader	4.0×10^{-6}	1.8×10^{-6}	7.2×10^{-8}	2.0×10^{-10}	1.6×10^{-8}	7.1×10^{-10}	1.4×10^{-9}
Observer	6.5×10^{-8}	3.1×10^{-8}	1.1×10^{-9}	3.4×10^{-12}	3.2×10^{-10}	1.3×10^{-11}	2.3×10^{-11}
Mechanical ground							
Applicator	3.6×10^{-6}	1.9×10^{-6}	2.2×10^{-7}	6.4×10^{-9}	1.5×10^{-7}	7.5×10^{-9}	2.7×10^{-9}
Mixer/loader	5.7×10^{-6}	2.9×10^{-6}	3.6×10^{-7}	1.1×10^{-8}	1.7×10^{-7}	8.7×10^{-9}	4.1×10^{-9}
Appl/mix/load	6.4×10^{-6}	3.3×10^{-6}	4.0×10^{-7}	1.2×10^{-8}	2.2×10^{-7}	1.1×10^{-8}	4.8×10^{-9}
Manual ground							
Backpack	5.6×10^{-5}	2.9×10^{-5}	4.7×10^{-6}	----	7.2×10^{-7}	1.4×10^{-8}	2.1×10^{-8}
Basal stem	----	1.4×10^{-6}	2.1×10^{-7}	1.2×10^{-8}	----	3.9×10^{-9}	----
Soil spot	----	----	----	----	----	----	----
Cut surface	2.5×10^{-5}	----	----	----	3.5×10^{-7}	----	7.0×10^{-9}

Note: Risks are upper 95 percent confidence limits.

used. As shown in the table, the exposures in 20 years of application work that lead to cancer risks greater than 1 in 1 million are backpack spraying of 2,4-DP and all mechanical and manual exposures to 2,4-D amine and ester formulations. Exposures to backpack sprayers using glyphosate result in a risk slightly higher than 1 in 1 million. The highest risk, greater than 5 in 100,000, is for backpack sprayer use of 2,4-D.

Cancer Risk From Brown-and-Burn Operations

The risk of cancer from exposure to herbicide residues in brown-and-burn operations was calculated assuming exposure of 6 hours per day, 20 days per year for 10 years. The results are given in table 5-27. The highest cancer risks from herbicides are 2 in 100 million for 2,4-D amine; 4 in 100 million for 2,4-D ester; 9 in 100 million for 2,4-DP; 1 in 10 billion for glyphosate; and 3 in 10 billion for picloram.

The risk of cancer from exposure to herbicide residues released from the burning of herbicide-treated vegetation can be put into perspective by comparing it with the risk of cancer from burning untreated woody vegetation, such as in a prescribed burn operation. When wood is burned, a variety of combustion products are formed. The types and relative abundance of these compounds varies with the temperature of the fire, the moisture content of the wood, and the species of wood. The two groups of compounds in wood smoke that are of greatest toxicological concern are polyaromatic hydrocarbons (PAH's) and the aldehydes. The PAH's in wood smoke include at least five chemicals that are carcinogens, including benzo(a)pyrene (BaP) and the aldehydes group, which includes formaldehyde, also a carcinogen. EPA has estimated a cancer potency for BaP of 0.0033 per (ug/m^3/day) (Haemisegger et al., 1985 in Dost, 1986). A cancer risk of 8.1×10^{-6}, approximately 8 in 1 million, was calculated for PAH's using methods by Dost (1986) and assuming 24 ug BaP/g of smoke particulate (based on measurements by White et al., 1985 in Dost, 1986); a smoke density of 30 mg/m^3; and 6 hours per day, 20 days per year, and 10 years of exposure. This risk is at least 90 times greater than the highest cancer risk from herbicide exposure during brown-and-burn operations.

In Region 8, workers are usually exposed, on the average, to brown-and-burn operations for 4 hours per day, 20 days per year, for 3 consecutive years. This would result in lower exposures to PAH's and cancer risks that are only one-fifth of those estimated by Dost (1986).

Comparison of Cancer Risks With Other Common Risks

To put the cancer risks calculated here in perspective, table 5-28 lists risks resulting from some more familiar hazards and occupational risks. Motor vehicle accidents have a risk of fatality that averages 2 in 10,000 per person each year. Over a 30-year period, the cumulative risk would be 6 in 1,000. A variety of hazards are listed in the table that have a risk of about 1 in 1 million. These hazards include smoking 2 cigarettes, eating 6 pounds of peanut butter, drinking 40 sodas sweetened with saccharin, or taking 1 transcontinental round trip by air. The cancer risk from a single x ray is 7 in 1 million. Many occupational risks are

Table 5-27

Cancer risk from brown-and-burn operations

Herbicide	Risk
2,4-D Amine	
Mechanical foliar-typical	6×10^{-9}
Mechanical foliar-maximum	2×10^{-8}
Foliar backpack/hand-typical	5×10^{-9}
Foliar backpack/hand-maximum	2×10^{-8}
Cut surface-typical	6×10^{-11}
Cut surface-maximum	2×10^{-8}
2,4-D Ester	
Mechanical foliar-typical	2×10^{-8}
Mechanical foliar-maximum	4×10^{-8}
Foliar backpack/hand-typical	1×10^{-8}
Foliar backpack/hand-maximum	2×10^{-8}
2,4-DP	
Mechanical foliar-typical	5×10^{-8}
Mechanical foliar-maximum	9×10^{-8}
Foliar backpack/hand-typical	6×10^{-9}
Foliar backpack/hand-maximum	2×10^{-8}
Glyphosate	
Aerial foliar-typical	3×10^{-11}
Aerial foliar-maximum	1×10^{-10}
Mechanical foliar-typical	8×10^{-14}
Mechanical foliar-maximum	7×10^{-12}
Foliar backpack/hand-typical	2×10^{-11}
Foliar backpack/hand-maximum	9×10^{-11}
Cut surface-typical	2×10^{-11}
Cut surface-maximum	1×10^{-10}
Picloram	
Mechanical foliar-typical	4×10^{-11}
Mechanical foliar-maximum	3×10^{-10}
Foliar backpack/hand-typical	3×10^{-12}
Foliar backpack/hand-maximum	2×10^{-11}
Cut surface-typical	8×10^{-15}
Cut surface-maximum	1×10^{-10}

Table 5-28

Lifetime risk of death or cancer[a] resulting from everyday activities

Activity	Need to Accumulate 1 in 1 Million Risk of Death	Average Annual Risk[b] per Capita
Based on living in the United States		
Motor vehicle accident	1.5 days	2 x [illegible][b]
Falls	6 days	6×10^{-5}
Drowning	10 days	4×10^{-5}
Fires	13 days	3×10^{-5}
Firearms	36 days	1×10^{-5}
Electrocution	2 months	5×10^{-6}
Tornados	20 months	6×10^{-7}
Floods	20 months	6×10^{-7}
Lightning	2 years	5×10^{-7}
Animal bite or sting	4 years	2×10^{-7}
Occupational Risks		
General		
Manufacturing	4.5 days	8×10^{-5}
Trade	7 days	5×10^{-5}
Service and government	3.5 days	1×10^{-4}
Transport and public utilities	1 day	4×10^{-4}
Agriculture	15 hours	6×10^{-4}
Construction	14 hours	6×10^{-4}
Mining and quarrying	9 hours	1×10^{-3}
Specific		
Coal mining (accidents)	14 hours	6×10^{-4}
Police duty	1.5 days	2×10^{-4}
Railroad employment	1.5 days	2×10^{-4}
Fire fighting	11 days	8×10^{-4}

Table 5-28 (continued)

Lifetime risk of death or cancer[a] resulting from everyday activities

Activity	Need to Accumulate a 1 in 1 Million Risk of Death	Average Annual Risk[b] per Capita
Everyday Risks		
Eating and drinking	40 diet sodas (saccharin)	NA
	6 pounds of peanut butter (aflatoxin)	NA
	180 pints of milk (aflatoxin)	NA
	200 gallons of drinking water from Miami or New Orleans	NA
	90 pounds of broiled steak (cancer risk only)	NA
Smoking	2 cigarettes	

[a] Cancer risks shown in this table were calculated based on a variety of assumptions that tend to overestimate risk as explained in section 5.

[b] To calculate the risk over a lifetime, multiply this column by 70. From Crouch and Wilson (1982).

NA: Not applicable.

greater. Working for 30 years in agriculture or construction has a risk of about 1.8 in 100, and in mining and quarrying the risk is even greater: 3 in 100 over 30 years.

RISK OF HERITABLE MUTATIONS

No human studies are available that associate any of the herbicides with heritable mutations. Furthermore, no risk assessments that quantify the probability of mutations are available in the literature or from EPA. Laboratory studies constitute the best available information on mutagenic potential. Results of the mutagenicity assays conducted on the 14 herbicides and additives are summarized in section 3 in table 3-3.

For some of the herbicides, no EPA-validated mutagenicity tests exist or the mutagenicity tests conducted are insufficient to conclude whether the chemical is mutagenic. For these herbicides, a very conservative assumption was to conclude that these herbicides have the potential to cause mutations in humans. In these cases the results of carcinogenicity tests (see table 3-3) or cancer risk assessments were used to give an indication of the risk of heritable mutations. The rationale for this assumption is summarized by the USDA (1985a) as follows:

> Since mutagenicity and carcinogenicity both follow similar mechanistic steps (at least those that involve genetic toxicity), the increased risk of cancer can be used to approximate the quantitative risk of heritable mutations. The basis for this assumption is that both mutagenicity and at least primary carcinogens react with DNA to form a mutation or DNA lesion affecting a particular gene or set of genes. The genetic lesions then require specific metabolic processes to occur, or the cells must divide to insert the lesion into the genetic code of the cell.
>
> We believe the cancer risk provides a worst case approximation to heritable mutations because cancer involves many types of cells whereas heritable mutations involve only germinal (reproductive) cells.

However, carcinogenic potency is not a completely reliable indicator of mutagenic potential. It is true that currently available data indicate that known carcinogens are likely to be mutagens, and known mutagens are likely to be carcinogens, but there are a significant number of exceptions that appear to be only carcinogens or only mutagens (Brusick, 1980). If the relationship between carcinogenicity and mutagenicity is not reliable, then quantitative estimation of mutagenic risk based on estimates of carcinogenic risk would be even more tenuous. Consequently, quantitative estimates of mutagenic risk will not be presented here.

Glyphosate, imazapyr, and sulfometuron methyl tested negative for mutagenicity in all assays conducted, and thus can be considered to pose negligible mutagenic risk.

Hexazinone, dicamba, picloram, tebuthiuron, and triclopyr were nonmutagenic in the majority of assays conducted and were nononcogenic in all of the carcinogenicity tests performed; therefore, it can be assumed that their mutagenic risk is slight to negligible.

Fosamine was negative for mutagenicity in four of five studies reported in EPA's summary of toxicity tests (EPA, 1987) and in a number of bioassays described in USDA (1984). Fosamine also has not been shown to cause cancer. Therefore, fosamine is considered to present a very low mutagenicity risk in this analysis.

No validated mutagenicity studies have been conducted with limonene. Limonene is a chemical that is "generally regarded as safe" by the Food and Drug Administration (see section 3), and it is not suspected of being mutagenic. However, to be conservative it is considered a possible mutagen in this risk assessment.

Studies on 2,4-D and on 2,4-DP have indicated both positive and negative mutagenic potential. EPA has requested more mutagenicity test information for both of these compounds. A number of comprehensive reviews of the 2,4-D mutagenic data have indicated that it does not pose significant risk of human gene mutations (USDA, 1984). The risk of heritable mutations from 2,4-D may be comparable to the estimates of cancer risk.

Mutagenic tests with 2,4-DP have shown mixed results. 2,4-DP was not mutagenic in four microbial assays but was mutagenic in four other assays; therefore, it may have limited genotoxic potential. Based on the limited test data presented in section 3, one cannot presume mutagenic hazard, because no *in vivo* or mammalian assays have been conducted. However, to be conservative, it may be assumed that 2,4-DP is mutagenic and the mutagenic risk may be comparable to the risk of cancer.

The majority of mutagenicity assays on diesel oil and kerosene were negative. However, both contain small amounts of the carcinogenic compounds benzene and benzo-a-pyrene. The risk of these light fuel oils causing heritable mutations should be very low, judging by the low risk of their causing cancer, as discussed previously.

RISK OF SYNERGISTIC AND CUMULATIVE EFFECTS AND EFFECTS ON SENSITIVE INDIVIDUALS

Synergistic Effects

Synergistic effects of chemicals are those that occur from exposure to two chemicals either simultaneously or within a relatively short period of time. Synergism occurs when the combined effects of two chemicals is greater than the sum of the effects of each agent given alone (simple additive effect). For example, a mixture of the herbicides 2,4-D and picloram has produced skin sensitization in test animals, while neither herbicide alone has been found to have this effect. Cigarette smoke and asbestos are both known carcinogens. When inhaled in combination, they have been found to increase cancer risk eightfold above the risk of persons inhaling asbestos who do not smoke.

Evidence of Synergistic Effects From Pesticides

However, instances of chemical combinations that cause synergistic effects are relatively rare. Kociba and Mullison (1985) in describing toxicological interactions with agricultural chemicals state:

> Our present scientific knowledge in toxicology indicates than an exposure to a mixture of pesticides is more likely to lead to additivity or antagonism rather than synergism when considering the toxicological effects of such a combination. To be conservative and for reasons of safety, an additive type of toxicological response is generally assumed rather than an antagonistic type of response.
>
> In the case of registered pesticides, a great amount of toxicological information is developed during the research and development of each individual pesticide. In addition to this information on individual pesticides, short term toxicity studies are always done prior to the selling of a pesticide mixture. Should synergism unexpectedly be present in a proposed commercial mixture of two pesticides, it would be identified in such cases and would then be dealt with accordingly. In toxicological tests involving a combination of commercial pesticides, synergism has generally not been observed.

The herbicide mixtures that are used in the Forest Service's program have not shown synergistic effects in humans. But, synergistic toxic effects of herbicide combinations other than EPA-registered commercial mixtures are not normally studied. Time and money normally limit toxicity testing to the effects of the herbicides individually. Combinations that could be tested are too numerous to make that testing feasible. Combinations of interest in this risk assessment include not only combinations of 2 or more of the 11 herbicides (there are 55 possible combinations of 11 herbicides taken 2 at a time), but also combinations of the herbicides with other chemicals, such as insecticides. Based on the limited amount of data available on pesticide combinations, it is possible but quite unlikely that synergistic effects could occur as a result of exposure to two or more of the herbicides considered in this analysis.

Likelihood of Exposure to Two Herbicides

It is highly unlikely that synergistic adverse effects could result from exposure to more than one herbicide applied in separate projects. There are several reasons for this. First, unlike the situation in conventional agriculture, herbicide residues in plants and soil are not expected to persist from one application to another, even for the more persistent herbicides.

Second, the 11 herbicides are known to be rapidly excreted from the body (see section 3). None of the herbicides has been found to accumulate in test animal body tissues, so exposure of an individual to two herbicides at different times would be unlikely to cause simultaneous residues within the body.

Third, public exposures to the herbicides should be low, except for accidents, and should occur only infrequently. The probability of an accidental exposure to any single herbicide is extremely low. Because the probability of a member of the public receiving a large exposure is so low for one herbicide, the probability of simultaneous large exposures to two herbicides is negligible. This is because the probability of two independent events occurring simultaneously is the product of the probabilities of the individual events. For example, if the probability of a person's receiving a certain exposure is 1 in 1,000 for each of two herbicides, the probability of receiving that exposure to both herbicides would be 1 in 1 million.

Risks From Herbicide Mixtures

Simultaneous exposure to more than one chemical is likely in cases where those chemicals are combined in a single spray mixture. Although most vegetation control projects in the EIS area would involve only a single herbicide, some areas would be treated with a mixture of herbicides, but only mixtures that have been approved for use by EPA.

The EPA guidelines for assessing the risk from exposures to chemical mixtures (EPA, 1986e) recommend using additivity models when little information exists on the toxicity of the mixture and when components of the mixture appear to induce the same toxic effect by the same mode of action. They suggest in their discussion of interactions (synergistic or antagonistic effects) of chemical mixtures that "There seems to be a consensus that for public health concerns regarding causative (toxic) agents, the additive model is more appropriate than any multiplicative model."

The EPA guidelines suggest using a hazard index, HI, as the model of additivity based on the dose and toxicity reference level (NOEL) for each chemical as follows:

$$HI = D_1/L_1 + D_2/L_2$$

where:

D_i is the dose of the i^{th} component and
L_i is the level of safety (NOEL)

As HI approaches 1, the risk from the mixture becomes greater and greater. On the basis of the highest exposures for workers in this risk assessment for systemic effects using the Weedone CB mixture of 2,4-D and 2,4-DP, the HI is 0.0040434. This amount shows little possibility of toxic effects. The inverse of this HI is 247, representing an MOS slightly lower than for the 2,4-D in the mixture alone.

Cumulative Effects

Cumulative effects are not likely to occur because none of the herbicides are persistent in the environment or in the human body, so no member of the public is likely to be chronically exposed through the Forest Service's program nor receive simultaneous exposures from these same herbicides used in any other programs.

There are instances when it could be argued that cumulative doses would occur. If an area is resprayed with an herbicide before herbicide from the previous spraying has been totally degraded, or if another use of the same herbicide occurs in the same area and overlaps its degradation in time, then it is possible for larger herbicide doses to occur than from a single application. Cumulative exposure also could occur in individuals who use one of the herbicides in their lawn or garden work or are exposed to an herbicide from nearby agricultural areas and are then exposed to the same herbicide as a result of the Forest Service application program.

Although herbicide doses from the other types of sources mentioned were not evaluated in the risk assessment, adverse health effects from cumulative doses in this program were analyzed. The total dose from various exposure routes estimated in this analysis should be greater than what a person would normally contact. This is because the assumptions in the risk assessment overestimate exposures from eating, drinking, and coming in contact with vegetation. To the extent that these estimates are large enough to cover exposure from other unknown sources, the risks from the hypothetical cumulative exposures should be no greater than the risks already discussed in this assessment.

Effects on Sensitive Individuals

Individual Sensitivity

Doull et al. (1980) describe "hypersensitivity" as the response of subjects at the lower end of the frequency distribution in a quantal dose-response curve. Quantal means a subject either exhibits the toxic response or does not, at a given dose level. If the response of a population of test animals to varying doses of a chemical follows a normal distribution (bell-shaped curve), the hypersensitive individuals are those on the left side of the curve that respond at much lower doses than the average. For example, if the average individual responds with toxic symptoms at a dose of 100 mg/kg and the standard deviation of the response is 30 mg/kg, about 95 percent of the individuals will have responded with those symptoms at doses from 40 to 160 mg/kg. More than 99 percent will have responded at doses from 10 to 190 mg/kg. Less than 0.15 percent of the population will have experienced toxicity at doses lower than 10 mg/kg. Applying this distribution of response to humans would mean that in a population of 10,000, fewer than 15 individuals would be likely to experience toxicity at doses lower than 10 mg/kg. Those 15 individuals could be considered the hypersensitive individuals in the population.

Although a safety factor of 10 has traditionally been used by regulatory agencies (NAS, 1977) to account for intraspecies (that is, interindividual) variation, Calabrese (1985) has shown that human susceptibility to toxic substances can vary by two to three orders of magnitude. Calabrese examined a number of studies of human responses to chemicals and found that the safety factor of 10 accounts for effects in 80 to 95 percent of a population.

Factors Affecting the Sensitivity of Individuals. Factors that may affect individual susceptibility to toxic substances include diet, age, heredity, preexisting diseases, and life style (Calabrese, 1978). These factors have been studied in detail for very few cases, and their significance in controlling the toxicity of the proposed herbicides is not known. However, enough data have been collected on other chemicals to show that these factors can be important.

Elements of the diet known to affect toxicity include vitamins and minerals. For example, the mineral selenium can prevent the destruction of blood-forming tissues by chronic heavy exposure to benzene. Large doses of vitamin C have also been shown to protect animals and humans from toxic effects of chronic benzene exposure. Vitamin A seems to have a preventative effect on cancer induced by chemicals such as benzo(a)pyrene (found in cigarette and wood smoke) and DMBA. This effect has been seen in laboratory animals and human epidemiological studies. The food additives BHT and BHA may also be active in preventing the carcinogenicity of benzo(a)pyrene. Various levels of the B vitamin riboflavin also have been tested with BaP with mixed results. Vitamin C has been shown to prevent nitrites from combining with amines to form nitrosamines, and vitamin E seems to be at least as effective. These vitamins would be likely to prevent formation of N-nitrosoatrazine and N-nitrosoglyphosate if conditions were otherwise favorable for their formation in the human stomach (Calabrese and Dorsey, 1984).

Genetic factors are also known in some cases to be important determinants of susceptibility to toxic environmental agents. Susceptibility to irritants and allergic sensitivity vary widely among individuals and are known to be largely dependent on genetic factors. Race has been shown to be a significant factor influencing sensitivity to irritants, and some investigations have indicated that women may be more sensitive than men (Calabrese, 1984).

Various human genetic conditions have been identified as possibly enhancing susceptibility to environmental agents. For example, persons with beta thalassemia may be at increased risk when exposed chronically to benzene. However, only one condition, G-6-PD deficiency, has been demonstrated conclusively to cause enhanced susceptibility to industrial pollutants. Several other genetic conditions have been shown to involve defects in the cellular mechanisms for repair of damage to DNA. Persons with these diseases share an increased sensitivity to the effects of UV light, which can cause cancer. Cells from individuals with at least one of these diseases, xeroderma pigmentosum, also are sensitive to a variety of chemical substances implicated as causative agents of human cancers. (Calabrese, 1984)

Persons with other types of preexisting medical conditions also may be at increased risk for toxic effects. For example, sensitivity to chemical skin irritants can be expected to be greater for people with a variety of chronic skin ailments. Patients with these conditions may be advised to avoid occupational exposure to irritating chemicals. (Shmunes, 1980, as cited in Calabrese, 1984)

Allergic Hypersensitivity

A particular form of sensitivity reaction to a foreign substance is allergic hypersensitivity. Allergic hypersensitive reactions may be immediate, such as in anaphylactic reactions to insect bites or penicillin injections; or they may be delayed as in the case of immune responses to tuberculin tests or contact dermatitis caused by poison ivy. The severe, immediate anaphylactic reactions, which can be fatal if not treated within minutes, are antigen-antibody reactions that require large, complex organic molecules to initiate the sensitivity. The delayed allergic hypersensitive reactions are usually directed against whole cells (bacteria, viruses, fungi) but, as in contact dermatitis, may be induced by lower molecular weight substances such as the catechols of poison ivy, cosmetics, drugs, or antibiotics. (Volk and Wheeler, 1983) Benzocaine, neomycin, formaldehyde, nickel, chromium, and thiram are all known to produce these reactions (Marzulli and Maibach, 1983).

Likelihood of Effects in Sensitive Individuals

Based on the current state of knowledge, individual susceptibility to the toxic effects of the 11 herbicides cannot be specifically predicted. As discussed above, safety factors have traditionally been used to account for variations in susceptibility among people. The margin-of-safety approach used in this risk assessment takes into account much of the variation in human response as discussed earlier by Calabrese (1985). As described in the introduction to this risk assessment, a safety factor of 10 is used for interspecies variation; an additional safety factor of 10 is used for within-species variation.

It is believed that the normal margin of safety of 100 for both types of variation is sufficient to ensure that most people will experience no toxic effects. However, unusually sensitive individuals may experience effects even when the margin of safety is equal to or greater than 100. In particular, in instances in the risk assessment where margins of safety are less than 100 for an exposure to a particular herbicide, it is possible that an exposed sensitive individual would experience toxic effects, whereas the average person would not. It must be noted, however, that in most applications that will actually occur when the program is implemented, no member of the public is likely to be exposed. Furthermore, because sensitive individuals constitute only a fraction of the population at large, it is highly unlikely that a sensitive individual would be exposed in any Forest Service application. It must also be noted that most public exposures that have been estimated to occur in this risk assessment are very low.

None of the herbicides in the Forest Service program is of high molecular weight, so the immediate allergic reactions and the delayed allergic reactions, except for contact dermatitis, are very unlikely as possible toxic effects. Some people may develop contact dermatitis from herbicide exposure, but this type of reaction would most likely be limited to workers who handle the herbicides regularly and are exposed to relatively large amounts on a number of occasions. The small, infrequent exposures of the public should limit the possibility of this type of reaction.

Section 6

WILDLIFE AND AQUATIC SPECIES HAZARD ANALYSIS

This section summarizes the toxicity of the herbicides proposed for use in Region 8 to wildlife and aquatic species. The term wildlife as used in this section refers to mammals, birds, reptiles, amphibians, and insects; aquatic species include fish, aquatic invertebrates, and aquatic life-stages of amphibians. Wildlife and aquatic species are discussed in separate subsections, each with an introduction that includes information on toxicity classifications and terminology. Common and scientific names for all species discussed are given at the end of section 8 in table 8-35.

WILDLIFE HAZARD ANALYSIS

This hazard analysis summarizes the findings of laboratory and field studies that indicate the toxicity to wildlife of the herbicides and additives proposed for use in Region 8. In many cases, laboratory studies of domestic animals have been used because of a lack of studies specifically on wildlife. The results of domestic animal studies are considered to be representative of the effects that would occur in similar species in the wild.

Differences in sensitivity to toxic substances that occur between species are primarily accounted for by differences in metabolism (Calabrese, 1983). Other important factors that also account for these differences in sensitivity are absorption, plasma protein binding, biliary excretion, and intestinal microflora (Calabrese, 1983).

Rodent toxicity studies, as well as carcinogenicity and mutagenicity results, have already been summarized in section 3, the Human Health Hazard Analysis. They will not be repeated in detail here. The relative toxicity of the chemicals, based on the range of LD_{50} values, was based on the same toxicity categories used by EPA for humans (see section 3). The toxicity rating used in this risk assessment for honey bees is that of Dr. Larry Atkins (University of California). It is based on the amount of herbicide required to kill a bee: less than 2 micrograms (ug)/bee is classified as highly toxic, 2 to 11 ug/bee is moderately toxic and greater than 11 ug/bee is relatively nontoxic (Al Vaughan, Ecological Effects Branch, Hazard Evaluation Division, EPA, personal communication, 1987).

The acute toxicity of the Region 8 herbicides and additives to rats and mallards is summarized in table 6-1.

2,4-D

2,4-D is moderately toxic to vertebrate species (table 6-2). There are significant differences in toxicity to vertebrates among the forms of 2,4-D (amines, butyl esters, isooctyl esters, and propylene glycol butyl ether esters) (Ghassemi et al., 1981). In many instances, toxic response to a specific 2,4-D formulation appears to be species-specific (USDA, 1984).

Table 6-1

Acute toxicity of Region 8 herbicides and additives to rats and mallard ducks

Herbicide/Additive	Oral LD_{50} (mg/kg) Rat	Oral LD_{50} (mg/kg) Mallard
2,4-D		
Acid	375	>2,000
Butyl ester	620	>2,025
2,4-DP	532	No data
Dicamba	757	>2,510 (Banvel)
Fosamine	24,400	>5,000
Glyphosate	4,320	>2,000[a]
Hexazinone	1,690	approx. 1,250[b]
Imazapyr	>5,000	>2,150
Kerosene	>28,000	No data
Diesel Oil	>7,380	16,400
Limonene	>5,000	No data
Picloram	8,200	>2,000
Sulfometuron methyl	>5,000	>5,000
Tebuthiuron	644	>2,000
Triclopyr technical	630	1,698
Garlon 3A (amine)	2,830	No data
Garlon 4 (ester)	2,140	>4,640

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[a] Bobwhite; no value for the mallard is available.

[b] Based on a dietary LC_{50} for mallards of 10,000 ppm and a conversion factor of 0.125 mg/kg/day per ppm in diet for chicks (Lehman, 1954).

Table 6-2

Acute oral toxicity of 2,4-D to mammals and birds

Species	Form of 2,4-D	LD_{50} (mg/kg)
Rat	Acid	375[a]
	Butyl ester	620[a]
Mouse	Acid	368[a]
	Butyl ester	380[a]
Guinea pig	Acid	469[a]
	Butyl ester	848[a]
Rabbit	Acid	800[a]
	Butyl ester	424[a]
Dog	Acid	100[a]
Cat	Butyl ester	820[a]
Cattle	Butyl ester	100[a]
Mule deer (8-11 months)	Acid	400 to 800[b]
Chicken	Acid	541[a]
	Butyl ester	2,000[a]
Mallard (3-5 months)	Acid	>2,000[b]
(4 months)	Amine (4 lb a.e./gal)	>2,025[b]
Pheasant (3-4 months)	Acid	472[b]
Pigeon	Acid	668[a]
Japanese quail (2 months)	Acid	668[b]
Chukar (4 months)	Acid	200 to 400[b]

[a] Source is USDA, 1984.
[b] Source is Hudson et al., 1984.

Oral LD_{50}'s in mammals range from 100 mg/kg for dogs, cattle, and swine to 848 mg/kg for guinea pigs (USDA, 1984; Ghassemi et al., 1981). Toxic effects include gastrointestinal disturbances, weight loss, muscle weakness, and loss of coordination (USDA, 1984). Mild to moderate eye, skin, and respiratory irritation is caused by some formulations (USDA, 1984). No teratogenic or reproductive effects have been observed in rats (EPA, 1986a).

In birds, acute oral LD_{50}'s range from 472 mg/kg in pheasants (3 to 4 months old) to more than 2,000 mg/kg in mallards (4 months old) (Hudson et al., 1984). Toxic effects include excessive thirst and salivation, tremors, exhaustion, and imbalance (Hudson et al., 1984). Eight-day dietary studies with the dimethylamine salt of 2,4 D and the butoxyethanol ester of 2,4-D yielded LC_{50} values of more than 5,000 ppm for Japanese quail, bobwhite quail, ring-necked pheasants, and mallard ducks (Hill et al., 1975, as cited in USDA, 1984). No reproductive or teratogenic effects were observed in the eggs of chickens and pheasants when sprayed with various forms of 2,4-D, even at dosage levels of up to 20 times the recommended field application rate (USDA, 1984). Chicken eggs injected with 2,4-D to give concentrations of 10, 50, 100, 200, and 300 ppm in the eggs resulted in hatching success rates of 83, 100, 71, 62 and 0 percent, respectively, of the control hatch (Dunachie and Fletcher, 1970, as cited in USDA, 1984). The LC_{50} of mallard eggs immersed in an aqueous emulsion of 2,4-D was a concentration equivalent to a field application rate of 215 kg/ha (192 lb/ac), which is 128 times the regional average field application rate of 1.68 kg/ha (1.5 lb/ac) (Hoffman and Albers, 1984).

The bioaccumulation ratio is low for tested animals exposed to 2,4-D, and accumulated residues are rapidly excreted once exposure ceases (Norris, 1981, as cited in USDA, 1984). Very few monitoring data exist on 2,4-D levels found in wildlife. However, studies by Erne (1974) in Sweden found levels of 2,4-D residues that ranged from 0.05 to 6 mg/kg in liver and kidney tissue of 250 samples of wildlife (including moose, roedeer, reindeer, red deer, fallow deer, hares, pheasants, grouse, and other species) taken by hunters or found dead during the period 1968 to 1972.

There is some indication in the literature that after treatment with 2,4-D, there is increased palatability (and possibly increased toxicity) of normally unpalatable weeds (Irvine et al., 1977). This was observed in ragwort (Senecio jacobaea, Britain's most serious poisonous weed to domestic livestock) after 2,4-D application (Irvine et al., 1977). Increased palatability was thought to be related to an increased water-soluble carbohydrate content. The authors reported that 2,4-D also may have increased the total unsaturated pyrrolizidine alkaloid content, thus increasing the plant's toxicity. Based on the results of this study, it was suggested that cattle be withheld from pastures for about 3 weeks after application of 2,4-D. Effects on grazing wildlife have not been reported.

Based on studies with honey bees, insects appear to be relatively tolerant to high levels of 2,4-D (USDA, 1984). The LD_{50} of 2,4-D for honey bees ranged from 11.525 ug/bee for an unspecified route of exposure to 105 ug/bee administered orally (USDA, 1984). Bees fed purified 2,4-D had decreased lifespans (approximately half the lifespan of bees exposed to lower doses) at 1,000 ppm; however, lifespans were not shortened in bees fed up to 1,000 ppm of the butoxyethanol ester, isooctyl ester, or the dimethylamine salt of 2,4-D (USDA, 1984). A temporary decrease in reproductive rate was observed in bees fed 100 ppm or more of an unspecified 2,4-D formulation (presumed to be an acid), although no effects were observed at 10 ppm. The effect was reversible and abated when exposure was stopped (USDA, 1984).

2,4-DP

Technical 2,4-DP is slightly toxic to mammals based on acute oral LD_{50}'s of 532 mg/kg in rats and 650 mg/kg in mice (EPA, 1984a). Technical 2,4-DP caused slight eye and dermal irritation in rabbits (EPA, 1984a). The acute oral LD_{50} for the Weedone formulation in rats is 2,200 mg/kg (EPA, 1984a). Toxic effects in rats in this study included depression, excessive salivation, and reduced motor activity and coordination. Weedone caused no dermal irritation and slight eye irritation in rabbits (EPA, 1984a). Technical 2,4-DP caused teratogenic effects in rabbits at 25 mg/kg, but caused no effects in rats at 100 mg/kg, the highest dose tested (EPA, 1984a). If 2,4-DP behaves similarly to 2,4-D, then animals would bioaccumulate 2,4-DP very slightly, and absorbed material would be rapidly excreted in its unmetabolized form (USDA, 1984).

Injection of 2,4-DP into chicken eggs caused reduced hatching at 100 ppm and complete inhibition of hatching at 200 ppm (Dunachie and Fletcher, 1970, as cited in USDA, 1984). No other toxicity data are available for birds. If the toxicity of 2,4-DP is similar to that of 2,4-D, then 2,4-DP would be of low toxicity to birds (USDA, 1984).

The toxicity of 2,4-DP to invertebrate species is expected to be similar to that of 2,4-D, which is slightly toxic to most insects (USDA, 1984).

Dicamba

Technical dicamba is slightly toxic to mammals based on oral LD_{50}'s of 757 mg/kg in rats and 1,189 mg/kg in mice (USDA, 1984). The oral LD_{50} for guinea pigs is 3,000 mg/kg and for rabbits is 2,000 mg/kg (HSDB, 1987a). Technical dicamba caused mild dermal irritation and mild to moderate eye irritation in rabbits (EPA, 1986b). The acute oral LD_{50} of the Banvel formulation is 1,707 mg/kg in rats (USDA, 1984). A study with Banvel showed that the chemical has a moderate potential for causing dermal sensitization in guinea pigs (EPA, 1986b). Ten daily oral doses of 250 mg/kg of the Banvel D formulation, or one oral dose of 1,000 mg/kg, caused no adverse effects in sheep (Palmer and Radeleff, 1969). However, two doses of 500 mg/kg of Banvel D caused death in sheep (Palmer and Radeleff, 1969). No toxicity studies with wildlife species have been reported.

Dicamba has not been observed to be teratogenic in rats and rabbits (EPA, 1986b). In a three-generation reproduction study with rats, no reproductive effects occurred at the highest dose tested, 25 mg/kg/day (EPA 1986b). Dicamba is rapidly excreted in urine, primarily in its parent form, although some is excreted either as a conjugate with glucoronic acid or as 3,6-dichloro-2-hydroxybenzoic acid, and dicamba does not bioaccumulate in animal tissues (USDA, 1984).

The Environmental Protection Agency (1983b) has characterized technical dicamba and formulated dicamba acid and its salts as slightly toxic to avian wildlife. The 8-day dietary LC_{50} of technical dicamba acid is greater than 10,000 ppm in both bobwhite quail and mallard ducks (EPA, 1983a). An acute oral LD_{50} of 673 mg/kg was reported for technical dicamba in pheasants (USDA, 1984). The acute oral LD_{50}'s of the formulated products were all greater than 2,510 mg/kg in mallards, and 8-day dietary LC_{50}'s were all greater than 4,640 ppm in mallards and bobwhite quail (EPA, 1983a). Results of avian toxicity studies on formulated products of dicamba are summarized in table 6-3.

No teratogenic effects were observed in chicken eggs injected with dicamba; however, hatching success was reduced at the highest dose tested of 400 ppm (USDA, 1984). The LC_{50} of mallard eggs immersed in an aqueous solution of dicamba was greater than a concentration equivalent to a field application rate of 200 lb/ac, which is more than 100 to 400 times the recommended field application level in Region 8 (Hoffman and Albers, 1984). However, eye malformations and stunted growth were observed at unspecified levels that were below the reported LC_{50} (Hoffman and Albers, 1984).

Most invertebrate studies indicate that dicamba is moderately toxic to insects. The oral LD_{50} of dicamba for honey bees ranged from 3.6 ug/bee to greater than 10 ug/bee (USDA, 1984). Contact studies with dicamba reported LD_{50}'s of greater than 100 ug/bee and greater than 91 ug/bee (2.6 percent mortality was observed at 91 ug/bee) (USDA, 1984). Such doses

Table 6-3

Results of avian toxicity studies with formulated dicamba

Formulation	Mallard	Bobwhite Quail
4 lb/gal dimethylamine salt (Banvel)	Oral LD_{50} >2,510 mg/kg Dietary LC_{50} >4,640 ppm	Dietary LC_{50} >4,640 ppm
1 lb/gal dimethylamine salt (Banvel CST)	Oral LD_{50} >2,510 mg/kg Dietary LC_{50} >5,620 ppm	Dietary LC_{50} >5,620 ppm
55% aluminum salt	Oral LD_{50} >2,510 mg/kg Dietary LC_{50} >5,620 ppm	Dietary LC_{50} >5,620 ppm
2 lb/gal sodium salt	Dietary LC_{50} >10,000 ppm	Dietary LC_{50} >10,000 ppm

Source: EPA, 1983a.

far exceed those encountered in the field because a field application of 1.12 kg/ha (1 lb/ac) would result in a contact dose equivalent to 1.25 ug/bee (Ghassemi et al., 1981). Ingestion of technical dicamba and the Banvel D4S formulation for up to 60 days had no effect on the mortality of honey bees at the highest dose tested of 1,000 ppm (Morton et al., 1972, as cited in USDA, 1984). Cockroaches fed 1,000 ppm dicamba in food showed no developmental or reproductive effects (USDA, 1984).

Based on current information, EPA (1983a) has concluded that dicamba is unlikely to directly affect wildlife species.

Fosamine

Based on acute oral LD_{50} values of 24,400 mg/kg in rats; 7,380 mg/kg in guinea pigs, and greater than 15,000 mg/kg in dogs for the Krenite formulation (41.5 percent active ingredient), fosamine is very slightly toxic to mammals (DuPont, 1983a; USDA, 1984). Krenite caused mild to moderate skin irritation and no eye irritation in rabbits (DuPont, 1983a). The acute oral LD_{50} of the Krenite S formulation (Krenite with surfactant added) is greater than 5,000 mg/kg in rats (DuPont, 1983a). Although Krenite S is not reported to be a dermal irritant, it is reported to be a moderate to severe eye irritant in rabbits (DuPont, 1983a). Sheep given Krenite in the diet for 90 days showed no adverse effects at doses of up to 2,500 ppm, the highest dose tested (Schneider and Kaplan, 1983, as cited in USDA, 1984). Unformulated fosamine and Krenite were not teratogenic in rats (USDA, 1984).

Rats administered 57 mg/kg of fosamine eliminated all of the dose within 72 hours (Chrzanowski et al., 1979). Approximately 87 percent of the dose was excreted in the feces and 13 percent in the urine. Thirteen percent of the eliminated dose had metabolized to carbamoylphosphonate acid, while the remainder was excreted unchanged. No toxicity studies with carbamoylphosphonate acid are available.

Unformulated fosamine is very slightly toxic to birds based on acute oral LD_{50}'s of greater than 5,000 mg/kg in mallard ducks and bobwhite quail (Schneider and Kaplan, 1983, as cited in USDA, 1984). The 8-day dietary LC_{50} of unformulated fosamine is greater than 10,000 ppm in mallards and bobwhite quail (Schneider and Kaplan, 1983, as cited in USDA, 1984). The acute oral LD_{50} of formulated fosamine is greater than 10,000 mg/kg in bobwhite quail and mallard ducks (DuPont, 1983a).

According to a study by Lutz-Ostertag (1983), the ammonium salt of fosamine (solutions of 1 to 5 percent) is teratogenic when sprayed directly onto fertilized eggs of quail and chickens; quail eggs are more frequently and severely affected. Teratogenic effects in the quail and chick embryos included slight to severe malformations. Embryotoxicity to these species was considered low (Lutz-Ostertag, 1983).

In a study recently submitted for publication by Dr. D. Hoffman of the U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, fertile bobwhite quail and mallard duck eggs submerged in 1.5-, 6.5-, and 30-percent fosamine solutions showed no teratogenic effects.

Embryotoxicity was observed at the higher concentrations. However, because the exposure method (submersion) and test concentrations greatly exaggerate the likely field exposures, fosamine is not considered hazardous to avian species (O'Neal, 1987).

Based on effects observed in honey bees, fosamine appears to be only slightly toxic to insects (USDA, 1984). The contact LC_{50} was greater than 10,000 ppm when bees were sprayed with a 42-percent formulation of fosamine ammonium salt (Schneider and Kaplan, 1983, as cited in USDA, 1984). The LD_{50} was greater than 200 ug/bee when fosamine was dissolved in solvent and applied directly to bees (O'Neal, 1987).

Glyphosate

Glyphosate is generally recognized to be of low toxicity in the environment (USDA, 1984). Acute oral LD_{50}'s are 4,320 mg/kg for the rat and 3,800 mg/kg for the rabbit (EPA, 1984b; USDA, 1984). Based on these values, glyphosate can be considered slightly toxic.

Oral LD_{50} values for the Roundup and Rodeo formulations in rats are 5,400 mg/kg and greater than 5,000 mg/kg, respectively (Monsanto, 1983, 1985). The oral LD_{50} of Roundup for goats is 4,860 mg/kg (Monsanto, 1985). Glyphosate, Roundup, and Rodeo are reported to be practically nonirritating or slightly irritating to the eyes and skin of rabbits (Monsanto, 1983, 1985). Based on a 26-month feeding study, a NOEL of greater than 31 mg/kg/day was established for rats (EPA, 1986c). In a 1-year oral study with dogs, a NOEL of 500 mg/kg/day (HDT) was determined (EPA, 1987). Glyphosate has caused no reproductive or teratogenic effects in rats or rabbits (EPA, 1984b).

Studies conducted on black-tailed deer in pens in the Pacific Northwest showed no gross adverse health effects caused by the use of glyphosate for vegetation management (Sullivan, 1985). Glyphosate-treated browse and commercial chow were as acceptable for consumption by deer as untreated food. Likewise, glyphosate-induced weed and shrub control did not adversely affect deer use of treated habitat areas for at least the first year after treatment.

In a study to evaluate the direct effects of glyphosate on small mammals, no adverse effects on reproduction, growth, or survival were observed in populations of deer mice during the year following treatment (Sullivan, 1985).

Glyphosate is slightly toxic to birds based on the acute oral LD_{50} of greater than 2,000 mg/kg in bobwhite quail (EPA, 1986d). The 8-day dietary LC_{50} is more than 4,000 ppm for both mallard ducks and bobwhite quail (EPA, 1986d). Avian reproduction studies yielded no reproductive effects at dietary exposure levels of up to 1,000 ppm (EPA, 1986d).

Residue and metabolism studies have indicated that glyphosate is incompletely absorbed across the gastrointestinal membranes and that in the vertebrates tested, there is minimal metabolism or retention by tissues and rapid elimination of residues (Monsanto, 1982).

Glyphosate is relatively nontoxic to insects based on the 48-hour acute toxicity of greater than 100 ug/bee in honey bees (EPA, 1986e).

Hexazinone

Based on toxicity data for birds and mammals, hexazinone presents a low hazard to wildlife species (EPA, 1982). The acute oral LD_{50} of technical hexazinone is 1,690 mg/kg in rats, 860 mg/kg in guinea pigs, and 2,258 mg/kg in bobwhite quail (EPA, 1984c; EPA, 1982). The acute oral LD_{50} of a 25-percent hexazinone solution is 6,887 mg/kg in rats (DuPont, 1984). The 8-day dietary LC_{50}'s of greater than 10,000 ppm for mallards and greater than 5,000 ppm for bobwhite quail indicate that technical hexazinone is practically nontoxic to birds (EPA, 1982). Formulated and unformulated hexazinone were irritating to the eyes but not to the skin of rabbits and guinea pigs (USDA, 1984; EPA, 1982). Hexazinone has not been observed to cause teratogenic or reproductive effects in rats or rabbits (EPA, 1984c; USDA, 1984). No appreciable bioaccumulation of hexazinone occurs in animal tissues (USDA, 1984). Hexazinone is readily metabolized and is rapidly excreted in the urine and feces of animals (USDA, 1984).

Hexazinone is relatively nontoxic to insects (DuPont, 1984). The LD_{50} of a topical application of a 90-percent soluble powder of hexazinone is greater than 60 ug/bee for honey bees (DuPont, 1984).

Imazapyr

Imazapyr is slightly toxic to mammals based on acute oral LD_{50}'s ranging from greater than 2,000 mg/kg in mice to greater than 5,000 mg/kg in rats (table 6-4) (EPA, 1985a; American Cyanamid Company, 1985). Technical imazapyr and the Arsenal formulation are reported to be irritating to the eyes and mildly irritating to the skin of rabbits but are reported as nonsensitizing to guinea pigs (EPA, 1985a; American Cyanamid Company, 1985). No teratogenic effects were observed in rats or rabbits (American Cyanamid Company, 1985). Imazapyr is rapidly eliminated in the urine and feces and does not appear to accumulate in animal tissues (EPA, 1985a).

Imazapyr is characterized by EPA (1985a) as practically nontoxic to avian species. Acute oral LD_{50}'s of technical imazapyr and the Arsenal formulation are greater than 2,150 mg/kg (HDT) in bobwhite quail and mallards (table 6-4) (American Cyanamid Company, 1984; EPA, 1985a). Dietary LC_{50}'s for formulated and unformulated imazapyr are greater than 5,000 ppm (HDT) for mallards and bobwhites (American Cyanamid Company, 1984). No adverse effects were observed at any of these doses.

Imazapyr appears to be relatively nontoxic to insects. The LD_{50}'s for honey bees of technical imazapyr are greater than 100 ug/bee (HDT), and the Arsenal formulation is greater than 25 ug/bee (HDT) (American Cyanamid Company, 1984). No effects were observed at either of these doses.

Light Fuel Oil

Kerosene and diesel oil are very slightly toxic to mammals based on the acute oral LD_{50}'s of greater than 28,000 mg/kg and 7,380 mg/kg,

Table 6-4

Acute oral toxicity of imazapyr to mammals and birds

Species	LD_{50} (mg/kg)
Rat	>5,000[a]
Mouse	>2,000[a]
Rabbit	>2,000[a]
Bobwhite quail	>2,150[b]
Mallard duck	>2,150[b]

[a] Source is American Cyanamid Company, 1985.
[b] Source is EPA, 1985a.

respectively, in rats (HSDB, 1987b; Beck et al., 1982). Toxic effects include loss of muscle coordination, nausea, languor, drowsiness, rapid heart beat, and shallow respiration (ITII, 1976). Diesel oil is extremely irritating to the skin of rabbits but nonirritating to the eyes (Beck et al., 1982). Kerosene is mildly irritating to the skin and eyes of rabbits and nonsensitizing in guinea pigs (Beck et al., 1982). Dermal exposure to 6,560 mg/kg of diesel oil for 3 weeks caused a 67-percent mortality rate in rabbits (API, 1982). Dermal exposure to kerosene for 28 days caused skin and liver lesions in rabbits at the highest dose tested of 2,000 mg/kg but not at the next highest dose of 1,000 mg/kg (API, 1983). Other adverse effects to the skin of the treated animals were observed at all three doses tested (200, 1,000, and 2,000 mg/kg), including cracking, scab formation, necrosis, and ulcerations (API, 1983). No teratogenic effects were observed in rats when exposed to kerosene and diesel vapors during gestation (Mecler and Beliles, 1979; Beliles and Mecler, 1982).

Diesel oil is very slightly toxic to birds when ingested based on the acute oral LD_{50} of greater than 16,400 mg/kg (greater than 20 ml/kg) in mallards (Hudson et al., 1984). The toxic effects included weakness, diarrhea, and regurgitation. However, diesel oil appears to cause adverse reproductive effects in birds. Traces of oil in a mallard's diet sharply reduce egg production (Biderman and Dury, 1980, as cited in U.S. Department of Energy, 1983). Application of only 1 microliter (ul) of No. 2 fuel oil on mallard eggs significantly reduced survival and hatchability (Szaro et al., 1978). In the same study, application of 5 ul reduced hatching success to 18 percent, and 20 ul killed all embryos. Similar toxicity was noted in pheasant eggs sprayed with diesel oil to runoff, which failed to hatch (Kopischke, 1972). Death appears to be related to the aromatic portion of the oil rather than the aliphatic portion (Szaro et al., 1978; Hoffman and Albers, 1984). In addition, oil carriers increase the toxicity of pesticides to eggs, apparently by increasing penetration through the shell and membrane (Hoffman and Albers, 1984).

Kerosene was not lethal when applied to mallard eggs at doses of 1 to 50 ul/egg (Hoffman and Albers, 1984). The low toxicity observed in this study was believed to be related to the lower aromatic hydrocarbon content of kerosene (Hoffman and Albers, 1984).

Diesel oil is highly toxic to insects based on high mortality of honey bees during the first 24 hours after spray treatment (Moffet et al., 1972). No information was available on the toxicity of kerosene to honey bees. Kerosene and diesel oil, when used as solvents or adjuvants, also have been observed to increase the toxicity of insecticides (Lagier et al., 1974; Tsuda and Okuno, 1985).

Limonene

Limonene is very slightly toxic to mammals based on the acute oral LD_{50} of greater than 5,000 mg/kg in rats (EPA, 1984d). The acute dermal LD_{50} is greater than 2,000 mg/kg in rabbits, and the acute inhalation LC_{50} is greater than 5 mg/l (= 5 ppm) in rats (JLB International Chemical, Inc., undated and 1983). Limonene is mildly irritating to the eyes and skin, and although inhalation is not harmful, it may cause dryness of the throat (JLB International Chemical, Inc., 1987). Ingestion may cause vomiting, nausea, and diarrhea.

EPA has approved the use of limonene for control of ticks and fleas on dogs and cats (Sheppard, 1987). No lesions or toxic signs were observed in cats dipped in a flea dip containing 78.2 percent limonene at the recommended concentration of 1.5 oz/gal (Hooser et al., 1986). At 5 to 15 times the recommended concentration, cats exhibited hypersalivation, incoordination, and tremors (Hooser et al., 1986).

No studies have been reported in which the toxicity of limonene to birds was evaluated.

According to Sheppard (1987), limonene is highly toxic to insects, including red imported fire ants, house flies, stable flies, black soldier flies, paper wasps, fleas, and gray crickets. Death is apparently caused by action on the nervous system.

Picloram

Picloram is slightly toxic to mammals, based on acute oral LD_{50}'s ranging from greater than 540 mg/kg in calves to 8,200 mg/kg in rats (table 6-5) (Lynn, 1965; Jackson, 1965). Technical picloram caused mild eye and skin irritation in rabbits (EPA, 1984e). Picloram was not teratogenic in rats at the highest does tested of 1,000 mg/kg (EPA, 1984e). In a study by John-Greene et al. (1985), picloram was not teratogenic in rabbits at 400 mg/kg (HDT). The Tordon 101 formulation caused no ill effects in sheep at single doses of 1,900 mg/kg, but it caused death at levels of 2,200

Table 6-5

Acute oral toxicity of picloram to mammals and birds

Species	LD_{50} (mg/kg)
Rat	8,200[a]
Mouse	2,000 to 4,000[a]
Rabbit	approx. 2,000[a]
Guinea pig	approx. 3,000[a]
Sheep	>720[b]
Calf	>540[b]
Chicken	approx. 6,000[a]
Mallard duck	>2,000[c]
Pheasant	>2,000[c]

[a] Source is Hudson et al., 1984.
[b] Source is Jackson, 1965.
[c] Source is Lynn, 1965.

mg/kg and above (Lynn, 1965). Temporary weight loss was the only adverse effect seen in calves given Tordon 101 in single doses of 1,900 to 3,163 mg/kg (Lynn, 1965). No toxic signs or adverse effects on growth were observed in sheep given 18 mg/kg/day of technical picloram in the diet for 33 days (Jackson, 1965). Stimulated growth and improved feed efficiency were observed in swine given 22 mg/kg of feed for an unspecified time (McCollister and Leng, 1969). Metabolic and residue studies in mammalian species indicate that picloram is rapidly eliminated unchanged in the urine following ingestion (USDA, 1984). No metabolites have been detected (USDA, 1984). In addition, picloram does not appear to accumulate to any significant extent in animal tissues (USDA, 1984).

Picloram is slightly toxic to birds based on LD_{50}'s that range from greater than 2,000 mg/kg in mallards and pheasants to approximately 6,000 mg/kg in chickens (table 6-5) (Lynn, 1965; Hudson et al., 1984). Regurgitation occurred shortly after mallards were treated, and pheasants exhibited tremors and mild decline of muscle coordination after treatment (Hudson et al., 1984). Subacute dietary LC_{50}'s for bobwhite and Japanese quail, ring-necked pheasants, and mallard ducks were all greater than 5,000 ppm (HSDB, 1987c). The 8-day dietary LC_{50} of the Tordon 101 formulation is greater than 10,000 ppm for bobwhite quail and mallard ducks (EPA, 1984e).

Japanese quail given 100 ppm in a 2-week dietary study showed no effects on feathering, reproduction, mortality, and weight (Kenaga, 1969). In a similar test at 1,000 ppm, egg fertility and hatchability were reduced the first week but not the second (Kenaga, 1969). A three-generation study with Japanese quail showed no effects on food consumption, reproduction, survival, and body weight when given 100, 500, or 1,000 ppm in the diet (Kenaga, 1969). In a 1-year study in which Japanese quail were given 100 ppm to 10,000 ppm in their diet, no effects on reproduction, feeding, or body weights were observed. Mortality rates of treated quail were lower than those of controls (Kenaga, 1969).

The LC_{50} of mallard eggs immersed in an aqueous emulsion of picloram was equivalent to a field application rate of 112 kg/ha (100 lb/acre), which is more than 10 times the recommended field application level (Hoffman and Albers, 1984). Spray treatment of fertile chicken eggs or ring-necked pheasant eggs with a dose equivalent to 2.8 kg/ha (2.5 lb/acre) of Tordon 101 did not affect embryonic development or subsequent growth of hatched chicks (EPA, 1984e).

Picloram is relatively nontoxic to insects based on an acute contact LD_{50} of greater than 14 ug/bee in honey bees (Kenaga, 1979). Honey bees given 1,000 ppm picloram in a 60-percent sucrose syrup showed no toxic effects after 14 days and no increase in mortality compared to the control group after 60 days (USDA, 1984).

Sulfometuron Methyl

Sulfometuron methyl is very slightly toxic to birds and mammals based on acute oral LD_{50}'s of greater than 5,000 mg/kg in the rat and mallard duck (EPA, 1984f; DuPont, 1983b). It is slightly irritating to rabbit eyes and skin but is nonsensitizing to guinea pigs (EPA, 1984f). No teratogenic effects have been observed in rats and rabbits exposed to sulfometuron methyl (EPA, 1984f); however, lower maternal body weights and decreased numbers of offspring were observed at 250 mg/kg/day in a reproduction study in rats (DuPont, 1986). The 8-day dietary LC_{50}'s are greater than 5,620 ppm in bobwhite quail and greater than 5,000 ppm in mallards (DuPont, 1983b). The LD_{50} was greater than 12.5 ug/bee when sulfometuron methyl was applied directly to bees (O'Neal, 1987). No other studies have been reported on the toxicity of sulfometuron methyl to wildlife or insect species.

Tebuthiuron

Tebuthiuron is moderately to slightly toxic to mammals and birds based on acute oral LD_{50}'s ranging from 186 mg/kg in rabbits to greater than 2,000 mg/kg in mallards and bobwhites (table 6-6) (EPA, 1986e; USDA, 1986). Tebuthiuron is slightly irritating to the eyes but not to the skin of rabbits (EPA, 1986e). It caused decreased body weight in weanling pups in

Table 6-6

Acute oral toxicity of tebuthiuron to mammals and birds

Species	LD_{50} (mg/kg)
Rat	644[a]
Mouse	579[b]
Rabbit	286[b]
Dog	>500[b]
Cat	>200[b]
Bobwhite quail	>2,000[b]
Mallard duck	>2,000[b]
Chicken	>500[b]

[a] Source is EPA, 1986e.
[b] Source is USDA, 1986.

a three-generation rat reproduction study at doses of approximately 20 mg/kg (LDT) (EPA, 1986e). In other studies, however, no teratogenic effects were observed in rats at doses of approximately 90 mg/kg (HDT) or in rabbits at 25 mg/kg (HDT) (EPA, 1986e). In subchronic oral toxicity studies, dogs experienced increased thyroid and spleen weight at 25 mg/kg (EPA, 1986e). Decreased body weight was observed in cattle at 100 ppm in a 162-day study (EPA, 1986e). Tebuthiuron was readily metabolized and eliminated in the urine of tested animals (USDA, 1986).

In subacute oral toxicity studies, doses of up to 1,500 ppm resulted in no deaths in mallards and bobwhites (Meyerhoff, 1981, as cited in USDA, 1986). In a 30-day oral study, chickens exhibited depressed growth at 2,500 ppm (EPA, 1986e). In 18-, 24-, and 27-week studies, no effects on growth, reproduction, or behavior were observed in bobwhite quail or mallard ducks when fed up to 100 ppm in the diet (Elanco Products Company, 1983, undated, as cited in USDA, 1986).

Honey bees sprayed with 30,000 ppm tebuthiuron, which is equivalent to 5.56 kg/ha (5 lb/ac), did not differ in survival from bees sprayed with water. Bees sprayed with 120,000 ppm, equivalent to 22.4 kg/ha (20 lb/ac), had significantly higher mortality than controls (USDA, 1984). Based on these results, tebuthiuron appears to be of relatively low toxicity to terrestrial invertebrates.

Triclopyr

Triclopyr is moderately toxic to mammals based on LD_{50} values that range from 310 mg/kg in guinea pigs to 729 mg/kg in male rats (table 6-7) (EPA, 1985b). Technical triclopyr is slightly irritating to the eyes and skin of rabbits (EPA, 1985b). The Garlon 3A and Garlon 4 formulations are slightly toxic, with oral LD_{50}'s of 2,830 and 2,140 mg/kg in rats (males and females, respectively) (Dow Chemical Company, undated). Garlon 3A may cause slight to moderate skin irritation and is moderately to severely irritating to eyes, and Garlon 4 may cause slight skin irritation but no eye irritation (Dow Chemical Company, undated). Ponies exposed to four daily doses of 60 mg/kg of triclopyr exhibited no adverse effects; however, exposure to four daily doses of 300 mg/kg caused depression, recumbency, decreased gastrointestinal activity, and respiratory and muscular distress (Osweiler, 1983).

No teratogenic effects have been observed in rabbits (EPA, 1985b) but a rat study reported fetotoxic effects at the lowest dose of 10 mg/kg/day (EPA 1986f). Triclopyr is rapidly excreted, primarily as the parent compound, through the kidneys in animals (USDA, 1984). Small quantities of two other compounds (the metabolite trichloropyridinol and a conjugated form of the parent, triclopyr acid) are also excreted (USDA, 1984). Triclopyr does not bioaccumulate in animal tissues in any significant amount (Dow Chemical Company, 1987).

Based on acute oral and dietary studies, triclopyr, Garlon 3A, and Garlon 4 are slightly toxic to birds (table 6-7). The acute oral LD_{50} of technical triclopyr is 1,698 mg/kg for mallard ducks, and the dietary LC_{50} ranges from 2,935 to greater than 5,000 ppm (Dow Chemical Company, undated; Kenaga, 1979). The dietary LC_{50}'s of Garlon 3A and Garlon 4 are all greater than 9,000 ppm (Dow Chemical Company, undated). A one-generation reproduction study showed no reproductive effects, symptoms of toxicity, or abnormal behavior when mallards were given up to 500 ppm in their diet for a 20-week period, including 10 weeks prior to egg laying and 10 weeks during egg laying (Dow Chemical Company, 1987). A similar study reported no reproductive or toxic effects in bobwhite quail exposed to dietary levels of up to 800 ppm for a 20-week period, including 11 weeks prior to egg laying and 8 weeks during egg laying (Dow Chemical Company, 1987).

The acute contact LD_{50} of triclopyr in honey bees is greater than 60 ug/bee, indicating that it is relatively nontoxic to insects (Kenaga, 1969). The contact LD_{50} for honey bees is greater than 100 ug/bee based on a 1985 study (Dow Chemical Company, 1985).

AQUATIC SPECIES HAZARD ANALYSIS

The toxicity to aquatic species of the herbicides and additives proposed for use in Region 8 is summarized in this section. Information is presented on the acute and chronic toxicities of the herbicides to fish,

Table 6-7

Acute toxicity of triclopyr to mammals and birds

Species	Formulation	Test	Results
Rat	Technical	Oral LD_{50}	729 mg/kg (male)[a] 630 mg/kg (female)[a]
Mouse	Technical	Oral LD_{50}	471 mg/kg[b]
Rabbit	Technical	Oral LD_{50}	550 mg/kg[b]
Guinea pig	Technical	Oral LD_{50}	310 mg/kg[b]
Rat	Garlon 3A	Oral LD_{50}	2,830 mg/kg (male)[a] 2,140 mg/kg (female)[a]
	Garlon 4	Oral LD_{50}	2,460 mg/kg (male)[a] 2,140 mg/kg (female)[a]
Mallard duck	Technical	Oral LD_{50}	1,698 mg/kg[c]
		Dietary LC_{50}	>5,640 ppm[d]
	Garlon 3A	Dietary LC_{50}	>10,000 ppm[a]
	Garlon 4	Oral LD_{50}	>4,640 mg/kg
		Dietary LC_{50}	>10,000 ppm[a]
Japanese quail	Technical	Dietary LC_{50}	3,278 ppm
Bobwhite quail	Technical	Dietary LC_{50}	2,935 ppm[c]
	Garlon 3A	Dietary LC_{50}	11,622 ppm[a]
	Garlon 4	Dietary LC_{50}	9,026 ppm[a]

[a] Source is Dow Chemical Company, undated.
[b] Source is EPA, 1985b.
[c] Source is Kenaga, 1979.
[d] Source is Dow Chemical Company, 1987.

aquatic invertebrates, and amphibians. The common and scientific names of aquatic species included in this hazard analysis are given in table 8-35 in section 8.

The relative acute toxicities of the herbicides are classified according to a scheme by EPA (1985c) where LC_{50} values are described as follows: <0.1 ppm (1 ppm = 1 mg/l), very highly toxic; 0.1 ppm to 1 ppm, highly toxic; >1 ppm to ≤10 ppm, moderately toxic; >10 ppm to ≤100 ppm, slightly toxic; and >100 ppm, practically nontoxic.

The information presented in this section is used in the Aquatic Risk Analysis section (in section 8) as a basis for selecting toxicity values for organisms representative of the aquatic environments in Region 8. In some cases, a number of toxicity tests have been conducted under various water quality conditions with a particular herbicide and a given species that have resulted in a range of LC_{50} values (for example, technical grade picloram and rainbow trout in Mayer and Ellersieck, 1986). In these cases, the lowest reported value from the range has been included in the table in the hazard analysis.

The terms listed below pertain to aquatic toxicology and are used frequently in this section:

LC_{50}--the concentration of a toxicant in water that is lethal to 50 percent of a population of test organisms within a specific period of time (usually reported for 96 hours).

EC_{50}--the concentration of a toxicant in water that has a specific effect on 50 percent of the test organisms. It is often used with animals where determining death is difficult, such as with Daphnia sp. In this case, immobilization of an animal is the measured endpoint.

MATC--maximum acceptable toxicant concentration, which is the hypothetical toxic threshold concentration of a toxicant in water bounded by the highest tested concentration that has no significant adverse effect and the lowest concentration having a significant effect.

Static test--toxicity tests (generally only acute tests) in which the solution in the test chamber is still (not flowing); the solution may be renewed during the course of the test.

Flow-through test--toxicity test (acute, subchronic, or chronic) in which the solution in the test chamber is flowing continuously or intermittently. Flow-through tests generally result in somewhat lower LC_{50}'s than static tests conducted under the same conditions.

2,4-D

The aquatic toxicity of the butoxyethanol ester of 2,4-D ranges from moderately to highly toxic (table 6-8). Acute LC_{50} values range from about 0.5 ppm to 10 ppm for most species. Amphipods and snails are among the most sensitive groups. Esters are typically 100 times more toxic than their corresponding acids and most amine formulations, but, in most cases, they rapidly hydrolyze to corresponding acids (Ghassemi et al., 1981). Bioaccumulation of 2,4-D is low, and it generally is rapidly excreted in the urine unchanged or as a conjugate (USDA, 1984). 2,4-D amine is practically nontoxic to amphibians (Johnson, 1976).

2,4-DP

Only a few aquatic toxicity studies are available for 2,4-DP; these are summarized in table 6-9. No studies are available for invertebrates or amphibians. No long-term studies are available for any aquatic species. A

Table 6-8

Toxicity of 2,4-D to aquatic organisms

Species	Concentration (ppm)	Effect	Source
	2,4-D amine		
Rainbow trout	1.0	Avoidance behavior	Folmar, 1976, 1978, as cited in USDA, 1984
	>100	96-hr LC_{50}	Mayer and Ellersieck, 1986
Chinook salmon	>100	96-hr LC_{50}	Mayer and Ellersieck, 1986
Green sunfish	25	No deaths after 8 days	Hiltibran, 1967, as cited in USDA, 1984
Bluegill	168 (123-230)[a]	96-hr LC_{50}	Mayer and Ellersieck, 1986
	40	No deaths at 12 days	Hiltibran, 1967, as cited in USDA, 1984
Smallmouth bass fry	236 (185-300)[a]	96-hr LC_{50}	Mayer and Ellersieck, 1986
	25	No deaths at 8 days	Hiltibran, 1967, as cited in USDA, 1984
Fathead minnow	335 (245-458)[a]	96-hr LC_{50}	Johnson and Finley, 1980
Channel catfish	119 (109-130)[a]	96-hr LC_{50}	Mayer and Ellersieck, 1986
Mosquitofish	405	96-hr LC_{50}	Johnson, 1978, as cited in USDA, 1984
Lake chubsucker	25	No deaths at 8 days	Hiltibran, 1967, as cited in USDA, 1984
Long-nosed killifish	15	No effect at 48 hours	Butler, 1965

Table 6-8 (continued)

Toxicity of 2,4-D to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Lymnodynastes peroni 1-week-old tadpoles	287	96-hr LC_{50}	Johnson, 1976
Giant Toad 1-week-old tadpoles	288	96-hr LC_{50}	Johnson, 1976
Crayfish	>100	48-hr LC_{50}	Sanders, 1970
Water flea	4.0 (3.4-4.9)[a]	48-hr EC_{50}	Mayer and Ellersieck, 1986
Seed shrimp	8.0 (5.9-10.8)[a]	48-hr EC_{50}	Mayer and Ellersieck, 1986
Scud *G. fasciatus*	>100	96-hr LC_{50}	Mayer and Ellersieck, 1986
Sowbug	>100	48-hr LC_{50}	Sanders, 1970, as cited in USDA, 1984
Eastern oyster	2.0	No effect at 96 hrs	Butler, 1965
Midge	>100	48-hr EC_{50}	Mayer and Ellersieck, 1986
Amphibia			
Adelotus brevis			
1-week-old tadpodes	200	96-hr LC_{50}	Johnson, 1976
4-week-old tadpoles	340	No deaths after 96-hours	
	2,4-D butoxyethanol ester		
Rainbow trout			
fingerlings	1.49	96-hr LC_{50}	Inglis and Davis, 1972
yearlings	10.0	96-hr LC_{50}	Dodson and Mayfield, 1979, as cited in USDA, 1986
Bluegill	1.2	96-hr LC_{50}	Mayer and Ellersieck, 1986

Table 6-8 (continued)

Toxicity of 2,4-D to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Fathead minnow	3.3	96-hr LC_{50}	Mayer and Ellersieck, 1986
Black bullhead	7.4	96-hr LC_{50}	Inglis and Davis, 1972
Crayfish	>100	48-hr LC_{50}	Sanders, 1970
Glass shrimp	1.4	48-hr LC_{50}	Sanders, 1970
Pink shrimp	1.0	48-hrs, no effect	
Water flea			
D. pulex	3.0	8 days, no effects	Sigmon, 1979, as cited in DEA, 1986
D. magna	5.6	48-hr LC_{50}	Sanders, 1970
Copepod	3.1	96-hr LC_{50}	Linden et al., 1979
Scud			
G. lacustris	0.44	96-hr LC_{50}	Sanders, 1969
G. fasciatus	5.9	96-hr LC_{50}	Sanders, 1970
Sowbug	2.6	96-hr LC_{50}	Mayer and Ellersieck, 1986
Seed shrimp	2.2	48-hr EC_{50}	Mayer and Ellersieck, 1986
	1.8	48-hr LC_{50}	Sanders, 1970
Stonefly			
Pteronarcys californica			
adult	>1000	96-hr LC_{50}	FWPCA, 1968, as cited in DEA, 1986
nymphs	1.6	96-hr LC_{50}	Sanders and Cope, 1968
Eastern oyster	3.75	96-hr EC_{50}, decrease in shell growth	Butler, 1965
Snail	0.32	at 6 wks 42% mortality	Lim, 1978, as cited in Halter, 1980

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[a] Range is for the 95% confidence interval.

Table 6-9

Toxicity of 2,4-DP[a] to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Bluegill			
adult	1.1	48-hr LC_{50}	Pimentel, 1971
fry	10	No deaths after 10 days	Hiltibran, 1967, as cited in USDA, 1984
juveniles	20[a]	No deaths after 12 days	Hiltibran, 1967, as cited in USDA, 1984
Lake chubsucker			
fry	1.5[a]	No deaths after 10 days	Hiltibran, 1967, as cited in USDA, 1984

[a] Granular 2,4-DP-isooctyl ester.

48-hour LC_{50} of 1.1 ppm (isooctylester) has been reported for adult bluegill (Pimentel, 1971). This value compares closely with the 96-hour LC_{50} of 1.2 ppm for the butoxyethanol ester of 2,4-D for the same fish species (table 6-8). Because of the close chemical similarities of the two herbicides, it is expected but not proven that their aquatic toxicities would be similar. In the absence of toxicity data for 2,4-DP, the aquatic toxicity reference values of 2,4-D will be used for estimating the hazard of 2,4-DP.

Dicamba

Dicamba is only slightly toxic to most aquatic organisms (table 6-10). The salts and free acid of dicamba are considered toxicologically equivalent because the salt hydrolyzes to the free acid in an aqueous environment (EPA, 1983b). Short-term LC_{50} values are greater than 10 ppm for fish, amphibia, and most invertebrates. The amphipod *Gammarus lacustris*, which has a 96-hour LC_{50} of 3.9 ppm, is more sensitive to dicamba than any other aquatic animal tested (Sanders, 1969). A 48-hour EC_{50} of 11 ppm was determined for *Daphnia pulex* (Sanders and Cope, 1966, as cited in Hulbert, 1975, as cited in USDA, 1984). *Daphnia magna*, with a 48-hour EC_{50} of greater than 100 ppm (Johnson and Finley, 1980) does not appear to be as sensitive as *D. pulex*. No long-term aquatic toxicity studies have been reported.

Fosamine

Fosamine is considered practically nontoxic to fish and invertebrates because all acute LC_{50} values are greater than 100 ppm (table 6-11).

Table 6-10

Toxicity of dicamba (88% technical) to aquatic organisms

Species	96-hour LC_{50} (ppm)	Source
Rainbow trout		
fingerlings (0.8 g)	28 135	Mayer and Ellersieck, 1986 Velsicol Chemical Corporation, as cited in Ghassemi et al., 1981
Cutthroat trout	>50	Woodward, 1982, as cited in USDA, 1984
Coho salmon		
juveniles	120[a]	Lorz et al., 1979, as cited in USDA, 1984
Bluegill		
fingerlings (0.9 g)	>50 135	Mayer and Ellersieck, 1986 Velsicol Chemical Corporation, 1979 as cited in Ghassemi et al., 1981
Glass shrimp	>56	Mayer and Ellersieck, 1986
Water flea		
Daphnia sp.	11[b]	Sanders and Cope, 1966, as cited in Hurlbert, 1975, as cited in USDA, 1984
D. magna (1st instar)	>100[b]	Mayer and Ellersieck, 1986
Scud		
G. fasciatus	>100	Mayer and Ellersieck, 1986
Sowbug	>100	Mayer and Ellersieck, 1986
Frog, tadpole		
Adelotus brevis (1-week old)	185	Johnson, 1976
Limnodynastes peroni (1-week old)	106	Johnson, 1976

[a] 48-hr LC_{50}.
[b] 48-hr EC_{50}.

Table 6-11

Toxicity of fosamine to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout			
adult	>100	96-hr LC_{50}	Mayer and Ellersieck, 1986
adult	>1,000[a]	96-hr LC_{50} (no effects at 1,000 ppm)	Schneider and Kaplan, 1983, as cited in USDA, 1984
yolk-sac fry (alevin)	367[b]	96-hr LC_{50}	USDA, 1984
eggs	1,456[b]	lowest 96-hr LC_{50}	USDA, 1984
Coho salmon	8,290[a]	96-hr LC_{50}	Schneider and Kaplan, 1983, as cited in USDA, 1984
	295[a]	96-hr EC_{50}, based on avoidance behavior; threshold at 8.9 ppm	Schneider and Kaplan, 1983, as cited in USDA, 1984
	198[a]	96-hr EC_{50}, acute stress based on leucocrit values; threshold at 4 ppm	Schneider and Kaplan, 1983, as cited in USDA, 1984
egg stage	25,377	lowest 96-hr LC_{50}	USDA, 1984
yolk sac fry (alevin)	618[b]	96-hr LC_{50}	USDA, 1984
fingerlings	2,669	lowest 96-hr LC_{50}	USDA, 1984
yearling	3,295	lowest 96-hr LC_{50}	USDA, 1984
Bluegill	670 (378-1,190)[a,c]	96-hr LC_{50}	Schneider and Kaplan, 1983, as cited in USDA, 1984
Fathead minnow	>1,000[a]	96-hr LC_{50} (no effects at 1,000 ppm)	Schneider and Kaplan, 1983, as cited in USDA, 1984
Channel catfish	>100	96-hr LC_{50}	Mayer and Ellersieck, 1986

Table 6-11 (continued)

Toxicity of fosamine to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Crayfish	3,547[b]	96-hr LC_{50}	DuPont, 1987 (unpublished, personal communication, Fred O'Neal, DuPont, Agricultural Products Department, Wilmington, Delaware, 1987)
Water flea			
D. magna	1,524 (1,310-1,720)[a,c]	48-hr LC_{50}	Schneider and Kaplan, 1983, as cited in USDA, 1984
Scud			
G. pseudolimnaeus	>100	96-hr LC_{50}	Mayer and Ellersieck, 1986
Midge	>100	48-hr LC_{50}	Mayer and Ellersieck, 1986

[a] Ammonium salt.
[b] Krenite (41.5% ammonium salt of fosamine).
[c] Range is for 95% confidence interval.

Yolk-sac fry, fingerlings, and eggs of salmonids are not acutely sensitive to fosamine (USDA, 1984). Ninety-six-hour EC_{50}'s based on avoidance behavior and white blood cell counts in coho salmon also are greater than 100 ppm (USDA, 1984). No toxicity studies with amphibians have been reported, and no long-term studies have been reported with aquatic organisms.

Glyphosate

Region 8 has proposed for use the following three formulations of glyphosate: Roundup, Rodeo, and Accord. Because of its surfactant content, Roundup is much more toxic to aquatic organisms than the other two formulations, which do not contain surfactants. Therefore, it is important to treat separately the risk of different formulations.

Roundup

The toxicity of the Roundup formulation (41 percent isopropylamine (IPA) salt of glyphosate, 15 percent surfactant, and 44 percent water) to aquatic organisms is summarized in table 6-12. Roundup is moderately to slightly toxic; most 96-hour LC_{50} values range from 2 to 18 ppm. The acute toxicity of Roundup is greater at pH 7.5 than pH 6.5, and toxicity also increases with increasing temperature (Folmar et al., 1979). Rainbow trout did not exhibit avoidance behavior at concentrations up to 10 ppm, whereas mayfly nymphs showed avoidance behavior at this level (Folmar et al., 1979).

Rainbow trout were exposed for 12 hours to 0.02, 0.2, and 2.0 ppm of formulated Roundup (Folmar et al., 1979). No effects were observed on fecundity or maturation of gonads after being held in freshwater for 30 days. Midge larvae also were exposed to 0.02, 0.2, and 2.0 ppm of Roundup. Significant increases in stream drift of the larvae were observed at the highest concentration.

Rodeo and Accord

The Rodeo formulation (53.5 percent isopropylamine salt of the active ingredient N-phosphonomethyl glycine and 46.5 percent water) of glyphosate is practically nontoxic to aquatic organisms (table 6-12). The 96-hour LC_{50}'s for fish are all greater than 1,000 ppm, and the 48-hour LC_{50} for *Daphnia magna* is 930 ppm (Monsanto, 1983). The toxicity of the Accord formulation (41.5 percent IPA salt and 58.5 percent water) is expected to be similar to Rodeo because both of the products have the same active ingredient and have water as the only inert ingredient.

Technical Glyphosate

Technical glyphosate is only slightly to practically nontoxic to fish and invertebrates (table 6-12). Studies with channel catfish, bluegill, rainbow trout, and largemouth bass indicate that glyphosate does not bioaccumulate in fish to any significant degree (Monsanto, undated). The toxicity of glyphosate or glyphosate-formulations to amphibians has not been reported in the literature.

An MATC of greater than 25.7 ppm has been reported in a long-term study with fathead minnows (Monsanto, undated). A 21-day study with *Daphina magna* determined a NOEL of 50 ppm based on decreased reproduction (Monsanto, undated).

Hexazinone

The aquatic toxicity of hexazinone is summarized in table 6-13. Hexazinone is practically nontoxic to fish; all 96-hour LC_{50}'s are greater than 100 ppm. EPA (1982, as cited in USDA, 1984) has described technical hexazinone as "practically nontoxic" to fish. It is slightly toxic to aquatic invertebrates (table 6-13). A 21-day NOEL of 10 ppm (technical) has been determined for *Daphnia* sp. (Mayack et al., 1982, and EPA, 1982, both as cited in USDA, 1984). No toxicity studies have been reported for amphibians. No chronic studies with aquatic organisms have been reported.

Table 6-12

Acute toxicity of Roundup, Rodeo, and technical glyphosate to aquatic organisms

Species	Concentration (ppm)	Effect	Source
	Rodeo		
Trout	>1,000	96-hr LC$_{50}$	Monsanto, 1983
	680-1,070[a]	96-hr LC$_{50}$	Mitchell et al. (in press)
Chinook salmon	750-1,440[a]	96-hr LC$_{50}$	Mitchell et al. (in press)
Coho salmon	600-1,000[a]	96-hr LC$_{50}$	Mitchell et al. (in press)
Bluegill	>1,000	96-hr LC$_{50}$	Monsanto, 1983
Carp	>10,000	96-hr LC$_{50}$	Monsanto, 1983
Water flea *D. magna*	930	48-hr LC$_{50}$	Monsanto, 1983
	Roundup		
Rainbow trout fingerlings (1 g)	1.3	96-hr LC$_{50}$	Folmar et al., 1979
fingerlings (2 g)	8.3	96-hr LC$_{50}$	Folmar et al., 1979
Chinook salmon	20	96-hr LC$_{50}$	Mitchell et al. (in press)
Coho salmon	22	96-hr LC$_{50}$	Mitchell et al. (in press)
Bluegill	5.0	96-hr LC$_{50}$	Folmar et al., 1979
	5.8	96-hr LC$_{50}$	Monsanto, 1985
Fathead minnow	2.3	96-hr LC$_{50}$	Folmar et al., 1979
	9.4	96-hr LC$_{50}$	Monsanto, 1985

Table 6-12 (continued)

Acute toxicity of Roundup, Rodeo, and technical glyphosate to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Channel catfish			
fingerlings (2.2 g)	13	96-hr LC_{50}	Folmar et al., 1979
swim-up fry	3.3	96-hr LC_{50}	Folmar et al., 1979
	16	96-hr LC_{50}	Monsanto, 1982
Grass carp	15	96-hr LC_{50}	Tooby et al., 1980
Carp	19.7	96-hr LC_{50}	Monsanto, 1982
Crayfish	>1,000	96-hr LC_{50}	Monsanto, 1982
Water flea			
D. magna	3.0	48-hr LC_{50}	Folmar et al., 1979
	5.3	48-hr LC_{50}	Monsanto, undated
Copepod	22	96-hr LC_{50}	Linden et al., 1979
Scud *G. pseudolimnaeus*	43	96-hr LC_{50}	Folmar et al., 1979
Mayfly nymphs	10	Avoidance behavior	Folmar et al., 1979
Midge larvae	18	48-hr EC_{50}	Folmar et al., 1979
	2	Significant increase in stream drift	Folmar et al., 1979
	Technical Glyphosate[b]		
Rainbow trout	140 (120-170)	96-hr LC_{50}	Folmar et al., 1979
	38	96-hr TL_{50}	USDA, 1981, as cited in USDA, 1984
	86	96-hr LC_{50}	Monsanto, 1985.

Table 6-12 (continued)

Acute toxicity of Roundup, Rodeo, and technical glyphosate to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Bluegill	140 (110-160)	96-hr LC_{50} (static test)	Folmar et al., 1979
	24	96-hr LC_{50} (flow-through test)	USDA, 1981, as cited in USDA, 1984
	78	96-hr TL_{50}	USDA, 1984
	120	96-hr TL_{50}	Monsanto, 1985b
Fathead minnow	97 (79-120)	96-hr LC_{50}	Folmar et al., 1979
	>25.7	MATC, no adverse effects on survival, growth, or reproduction during 255 days of exposure	Monsanto, 1985
Channel catfish	130 (110-160)	96-hr LC_{50}	Folmar et al., 1979[a]
Carp	115	96-hr LC_{50}	USDA, 1981, as cited in USDA, 1984
Water flea			
Daphnia sp.	780	48-hr LC_{50}	Monsanto, 1985
D. magna	50	NOEL, based on reduced reproduction at 96 ppm; 21 days of exposure	Monsanto (undated)
Midge	55	48-hr EC_{50}	Folmar et al., 1979

[a] Combined with X-77 surfactant.

[b] Technical glyphosate (95% or more of active ingredient) is assumed to be the formulation used.

Table 6-13

Toxicity of hexazinone to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	320-420[a]	96-hr LC_{50}	EPA, 1982, as cited in USDA, 1984; Mayer and Ellersieck, 1986
	>180[b]	96-hr LC_{50}	
Brook trout	>100[a,b]	96-hr LC_{50}	Mayer and Ellersieck, 1986
Bluegill	505[c] (450-538)	96-hr LC_{50}	EPA, 1982, as cited in USDA, 1984
	370-420	96-hr LC_{50}	EPA, 1982, as cited in USDA, 1984
	925 (782-1,049)[c,d]	96-hr LC_{50}	Schneider and Kaplan, 1983, as cited in USDA, 1984
Fathead minnow	274 (207-361)[b,c]	96-hr LC_{50}	EPA, 1982, in USDA, 1984
Fiddler crab	>1,000[b]	96-hr LC_{50}	EPA, 1982, as cited in USDA, 1984
Grass shrimp	56-100[b]	96-hr LC_{50}	EPA, 1982, as cited in USDA, 1984
Water flea *Daphnia* sp.	20-50[b]	21-day LC_{50}	Mayack et al., 1982, as cited in USDA, 1984; EPA, 1982, as cited in USDA, 1984
	10[b]	21-day NOEL	
D. magna	151.6 (125.2-172.8)[b,c]	48-hr LC_{50}	EPA, 1982, as cited in USDA, 1984

Table 6-13 (continued)

Toxicity of hexazinone to aquatic organisms

Organism	Concentration (ppm)	Effect	Source
Aquatic invertebrates	.006-.044	Intermittent exposure, field sampling over 8 months indicated no major alterations in species composition or species diversity	Mayack et al., 1982, as cited in USDA, 1984
Eastern oyster larvae	320-560[b]	48-hr EC_{50}, based on reduction in number of normal embryos	EPA, 1982, as cited in USDA, 1984

[a] Technical.
[b] 90% wettable powder.
[c] Range is 95% confidence interval.
[d] Velpar L, 25% hexazinone liquid.

Imazapyr

Technical imazapyr, the isopropylamine salt of imazapyr, and the Arsenal 2.0 AS formulation are practically nontoxic to rainbow trout, bluegill, and channel catfish (table 6-14). The water flea, the only aquatic invertebrate that has been tested, was not sensitive to Arsenal (American Cyanamid Company, 1985). No studies have been reported with amphibians. Chronic or reproductive studies have not been reported in the literature.

Light Fuel Oil

Diesel fuel, jet fuels, and fuel oils are moderately to highly toxic to fish (table 6-15). Jenkins et al. (1977, as cited in Burks, 1982) studied the acute and chronic toxicity of jet fuels to several fish species, including the Golden shiner, rainbow trout, and flagfish. The 96-hour LC_{50}'s (static tests) for the Golden Shiner were 0.68 and 0.94 ppm for the jet fuels RJ-4 (a 12-carbon molecule) and RJ-5 (a 14-carbon molecule), respectively. The 97-day nonlethal concentration for rainbow trout was less than 0.03 ppm for RJ-4 and 0.04 ppm for RJ-5. The NOEL for eggs of the flagfish exposed by continuous flow to RJ-4 was 0.2 ppm. Reduced hatchability of flagfish was observed from exposure to RJ-5 at concentrations above 0.05 ppm.

Table 6-14

Toxicity of imazapyr to aquatic organisms

Species	Concentration (ppm)	Effect
Rainbow trout	110[a] >100[b]	96-hr LC_{50}
Bluegill	>180[a] >100[b] >1,000[c]	96-hr LC_{50}
Channel catfish	>100[b]	96-hr LC_{50}
Water flea *D. magna*	>350[a] 100[b] 750[c]	48-hr LC_{50}

[a] Arsenal 2.0 AS.
[b] Technical imazapyr.
[c] Isopropylamine salt of imazapyr.

Source: American Cyanamid Company, 1985.

Acute toxicity values (96-hour LC_{50}'s) for freshwater fish of greater than 0.19 ppm for diesel fuel and greater than 1.2 ppm for No. 2 fuel oil have been reported by EPA (1976, as cited in DOE, 1983). Tagatz (1961, as cited in Burks, 1982) reported a 48-hour LC50 for No. 2 fuel oil of 125 to 251 ppm with juvenile American shad. This reported concentration is based on the amount of oil applied to the water's surface (nominal concentration) and not the water-soluble fraction. This may account for the apparent lower sensitivity of the shad to No. 2 fuel oil.

The toxicity of No. 2 fuel oil has been studied for a number of marine fish and invertebrate species (table 6-15). The LC_{50}'s range from 0.81 to greater than 6.9 ppm for marine fish and 0.21 to 14.1 ppm for invertebrates (Connell and Miller, 1984). The range of toxicity values determined for No. 2 fuel oil with marine species is useful in estimating the range of sensitivities for freshwater species because marine and freshwater species generally have a similar range of tolerance to toxicants (Sprague, 1985).

Irwin (1964, as cited in Burks, 1982) calculated a "ratio of resistance" to rank the sensitivities of 57 fish species to oil refinery wastewater. The guppy was the least sensitive and was assigned a ratio of resistance of 100. The ratios of resistance for some common freshwater fish were as

Table 6-15

Toxicity of light fuel oil to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Freshwater fish	>0.19[a] >1.2[d]	96-hr LC_{50} 96-hr LC_{50}	EPA, 1976, as cited in DOE, 1983
Rainbow trout	<0.03[b] 0.04[c]	97-day nonlethal level 97-day nonlethal level	Jenkins et al., 1977, as cited in Burks, 1982
Dolly Varden trout smolts	2.29[d]	96-hr LC_{50}	Connell and Miller, 1984
Pink salmon	0.81[d]	96-hr LC_{50}	Connell and Miller, 1984
Golden shiner	0.68[b] 0.94[c]	96-hr LC_{50} 96-hr LC_{50}	Jenkins et al., 1977, as cited in Burks, 1982
Sheepshead minnow	>6.9[d]	96-hr LC_{50}	Connell and Miller, 1984
Saffron cod	2.93[d]	96-hr LC_{50}	Connell and Miller, 1984
Flagfish (eggs)	0.2[b] >0.05[c]	No effect level Reduced hatchability	Jenkins et al., 1977, as cited in Burks, 1982
Blue crab	14.1[d]	96-hr LC_{50}	Melzian, 1983
Grass shrimp		96-hr LC_{50}	Connell and Miller, 1984
larvae	1.2[d]		
post larvae	2.4[d]		
adult	3.5[d]		
Brown shrimp		96-hr LC_{50}	Connell and Miller, 1984
late juvenile	2.9[d]		
adult	4.9[d]		

Table 6-15 (continued)

Toxicity of light fuel oil to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Dark shrimp	1.11[d]	96-hr LC_{50}	Connell and Miller, 1984
Humpback shrimp	1.69[d]	96-hr LC_{50}	Connell and Miller, 1984
Scooter shrimp	0.53[d]	96-hr LC_{50}	Connell and Miller, 1984
Pink shrimp	0.21[d]	96-hr LC_{50}	Connell and Miller, 1984
Polychaete (segmented aquatic worm)	2-4.2[d]	96-hr LC_{50}	Connell and Miller, 1984

[a] Diesel fuel.
[b] Jet fuel RJ-4.
[c] Jet fuel RJ-5.
[d] No. 2 fuel oil.

follows: rainbow trout (34.68), smallmouth bass (35.60), northern pike (37.31), fathead minnow (49.19), largemouth bass (53.27), bluegill (54.10), and channel catfish (60.15). This study may be useful in predicting the relative order of sensitivities of these species to diesel fuels and other petroleum products.

The 96-hour LC_{50} for adult blue crabs exposed to No. 2 fuel oil was 14.1 ppm (Melzian, 1983). This species appears to be much more tolerant than other crustaceans or fish tested. No histopathological changes were observed in the gills, hepatopancreas, or muscles of the blue crab after 2 weeks of exposure to No. 2 fuel oil at 0 to 1.0 ppm (Melzian, 1983).

A spill of No. 2 fuel oil into a small stream in Virginia was acutely toxic to some fish, crayfish, and caddis flies (order Trichoptera) (Hoehn et al., 1974, as cited in Burks, 1982). Two weeks after the spill, the density of benthic macroinvertebrates downstream was 25 percent less than the density upstream from the spill, but species diversity was not affected. The density of the macroinvertebrates returned to normal levels by 18 weeks after the spill.

The toxicity of diesel fuel or other related petroleum compounds to amphibians has not been reported in the literature. No chronic toxicity studies have been reported for any aquatic organisms.

Limonene

Very little information is available on the toxicity of limonene (Cide-kick) to fish or other aquatic species. Watkins and Thayer (1982) have indicated that Cide-kick is moderately toxic to bluegills with a 96-hour LC_{50} of 5.2 (4.8-5.6) ppm. No information is available on its toxicity to aquatic invertebrates or amphibians. No long-term studies of the effects on any aquatic organism have been reported.

Picloram

Tordon 101 (a mixture of picloram and 2,4-D) is slightly toxic, and picloram is generally moderately to slightly toxic to aquatic organisms. All reported LC_{50}'s for Tordon 101 are greater than 10 ppm (table 6-16).

Aquatic insects and crustaceans have 24- to 96-hour LC_{50}'s of greater than 25 ppm for technical picloram. A 48-hour LC_{50} of 50.7 ppm has been reported for *Daphnia magna* exposed to technical picloram (Mayes and Dill, 1984). *Daphnia* sp. showed no effect during a 24-hour exposure to 380 ppm of Tordon 101 (USDA, 1984). For lake trout and cutthroat trout, technical grade picloram (90-percent active ingredient) is more toxic than the other formulations, with 96-hour LC_{50}'s in these species of 4.3 and 4.8 ppm, respectively (Johnson and Finley, 1980).

Woodward (1979) reported increased fry mortality in cutthroat trout at concentrations of picloram (technical grade) greater than 1.3 ppm and reduced fry growth above 0.61 ppm (flow-through tests). No adverse effects to cutthroat fry occurred below 0.29 ppm. The reported concentrations are initial peak concentrations, which are intended to simulate concentration resulting from runoff from a rainstorm. Mean concentrations for the exposure period were not reported. Similar findings have been reported by Scott et al. (1977, as cited in Mullison, 1985). Woodward (1976) has also reported chronic studies on lake trout, where 0.035 ppm of picloram adversely affected the rate of yolk sac absorption and growth of fry.

Mayes et al. (1987) conducted chronic toxicity studies with embryo-larval rainbow trout exposed to technical picloram. They reported an MATC of between 0.55 ppm and 0.88 ppm and estimated as 0.70 ppm based on the geometric mean. Larval survival was significantly reduced at 2.02 ppm, and growth was significantly reduced at 0.88 ppm.

No adverse effects on growth were reported for algae, *Daphnia* sp., goldfish, and guppies exposed to 1 ppm picloram for 10 weeks. Guppies exhibited no adverse effects at this same concentration after 6 months of exposure (Lynn, 1965, as cited in Ghassemi et al., 1981). Chronic studies with *Daphnia magna* by Gersich et al. (1985) indicated an MATC of between 11.8 an 18.1 ppm with a geometric mean of 14.6 ppm. The MATC endpoint was based on mean total young/adult.

Table 6-16

Toxicity of picloram to aquatic organisms

Species	Concentration (ppm)	Effect	Source
	Tordon 101[a]		
Rainbow trout	40.4	96-hr LC_{50}	Lynn, 1965; Winston, 1963, as cited in Kenaga, 1969
Brook trout	64.6	96-hr LC_{50}	Lynn, 1965; Winston, 1963, as cited in Kenaga, 1969
Brown trout	61.9	96-hr LC_{50}	Lynn, 1965; Winston, 1963, as cited in Kenaga, 1969
Coho salmon	17.5	24-hr LC_{50}	Spehar et al., 1981[a], as cited in USDA, 1984
Green sunfish	40.4	96-hr LC_{50}	Kenaga, 1969
Fathead minnow	17.4	96-hr LC_{50}	Lynn, 1965; Winston, 1963, as cited in Kenaga, 1969
Pugnose minnow	35.8	96-hr LC_{50}	Kenaga, 1969
Goldfish	20.2	24-hr LC_{50}	Hardy, 1963, as cited in Kenaga, 1969
Amphibia 1-week old tadpoles			
Adelotus brevis	95[b]	96-hr LC_{50}	Johnson, 1976
Limnodynastes peroni	105[b]	96-hr LC_{50}	Johnson, 1976
Water flea			
Daphnia sp.	530	95% mortality at 24 hr; no mortality at 380 ppm	Lynn, 1965
Snail	530	100% mortality at 72 hr; no mortality at 380 ppm	Lynn, 1965

Table 6-16 (continued)

Toxicity of picloram to aquatic organisms

Species	Concentration (ppm)	Effect	Source
	Picloram		
Rainbow trout	24-34	24 to 96 hr LC_{50}	U.S. DOI, 1965, as cited in Kenaga, 1969
Coho salmon	21-29	96-hr LC_{50}	Bond et al., 1967, as cited in Kenaga, 1969
Bluegill	21-26.5	96-hr LC_{50}	Bond et al., 1967, as cited in Kenaga, 1969
Largemouth bass	13.1-19.7	24 to 48-hr LC_{50}	U.S. DOI, 1964, as cited in Kenaga, 1969
Goldfish	14-36	24 to 96-hr LC_{50}	U.S. DOI, 1964, as cited in Kenaga 1969
Mosquito fish	120-133	24 to 96-hr LC_{50}	Johnson, 1978, as cited in USDA, 1984
Brown shrimp	1	48-hr NOEL	U.S. DOI, 1966, as cited in USDA, 1984
Water flea *Daphnia sp.*	530	95-percent mortality at 24 hours, NOEL at 380 ppm	Lynn, 1965
	1	No observed effect on growth and reproduction after 10 weeks	Hardy, 1966, as cited in USDA, 1984
Scud *G. lacustris*	48	48-hr LC_{50}	U.S. DOI, 1968, as cited in USDA, 1984

Table 6-16 (continued)

Toxicity of picloram to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Eastern oyster	1	No observed effect on shell growth after 48 hours	Butler, 1965
		Technical Grade (90% a.i.)	
Rainbow trout	3.1	96-hr LC_{50}, toxicity greater in hard water	Mayer and Ellersieck, 1986
	0.70	MATC, reduced growth in embryo larvae	Mayes et al., 1987
Lake trout	1.6	96-hr LC_{50}, toxicity greater in hard water	Mayer and Ellersieck, 1986
	0.035	Decreased rate of yolk sac absorption and growth in fry, chronic exposure	Woodward, 1976
Cutthroat trout	1.5	96-hr LC_{50}	Mayer and Ellersieck, 1986
	>1.3	After 22 days exposure, increased fry mortality;	Woodward, 1979
	>0.610	reduced growth of fry;	
	<0.29	no adverse effects	
Bluegill	13.5	96-hr LC_{50}, toxicity greater in hard water	Mayer and Ellersieck, 1986
Channel catfish	1.4	96-hr LC_{50}	Mayer and Ellersieck, 1986
Water flea	50.7	48-hr LC_{50}	Mayes and Dill, 1984
	68.3	48-hr LC_{50}	Gersich et al., 1985
	14.6	MATC based on mean total young per adult	Gersich et al., 1985
Scud *G. lacustris*	27	96-hr LC_{50}	Sanders, 1969

Table 6-16 (continued)

Toxicity of picloram to aquatic organisms

Organism	Concentration (ppm)	Effect	Source
Stoneflies			
Pteronarcella badia	>10.0	96-hr LC_{50}	Mayer and Ellersieck, 1986
Pteronarcys californica	48	96-hr LC_{50}	Sanders and Cope, 1968

[a] 10.2% picloram-triisopropylaniene salt, 5.7% a.e., and 21.2% a.e. 2,4-D triisopropylaniene salt).
[b] Tordon 50-D.

Studies with picloram (Tordon 50-D) have reported 96-hour LC_{50}'s for 1-week-old tadpoles of 95 ppm for *Adelotus brevis* and 105 ppm for *Limnodynastes peroni* (Johnson, 1976).

Sulfometuron Methyl

Acute toxicity tests using technical sulfometuron methyl were conducted with representative aquatic species, including bluegill, rainbow trout, crayfish, and *Daphnia magna* (table 6-17). The results indicate that this herbicide is only slightly toxic to aquatic organisms.

The fathead minnow was used for early lifestage aquatic toxicity testing. No effect on embryo hatch or larval survival and growth was observed at concentrations of up to 1.2 mg/l (DuPont, 1983b).

The toxicity of sulfometuron methyl to amphibians has not been reported in the literature. No long-term studies of the effects of sulfometuron methyl on aquatic organisms have been reported.

Tebuthiuron

The toxicity of tebuthiuron to aquatic organisms is summarized in table 6-18. This herbicide is practically nontoxic to most fish and invertebrates. Acute toxicity values are greater than 100 ppm for all aquatic species tested with the exception of the pink shrimp (96-hour LC_{50} = 48 ppm). Based on early life stage studies, NOEL's of 26 ppm been determined for rainbow trout, 9.3 ppm for fathead minnow, and 21.8 ppm for *Daphnia magna*. No studies are available on amphibians.

Triclopyr

The toxicity of triclopyr to aquatic species is summarized in table 6-19. The butoxyethyl ester is highly toxic to fish, whereas the triethylamine (TEA) salt is practically nontoxic. The 96-hour LC_{50} for bluegill exposed to the butoxyethyl ester is 0.87 ppm and is 891 ppm for exposure to the triethylamine salt. Unformulated triclopyr also is practically nontoxic to aquatic organisms. An 8-day embryo-larval study with fathead minnows exposed to the TEA salt formulation determined an MATC of 91 ppm based on mortality (Mayes et al., 1984). The hatchability of the embryos, development, and growth of the fry were not significantly affected. No toxicity studies have been reported with amphibians.

Table 6-17

Toxicity of sulfometuron methyl to aquatic organisms

Species	Concentration (ppm)	Effect
Rainbow trout	$>12.5^{a}$	96-hr LC_{50}
Bluegill	$>12.5^{a}$	96-hr LC_{50}
Fathead minnow	1.2	No effect on eggs or larvae
Crayfish	$>5{,}000^{b}$	96-hr LC_{50}
Water flea		
D. magna	$>12.5^{a}$ $8{,}500^{c}$	48-hr LC_{50} 48-hr EC_{50}

[a] This represents the limits of solubility for the technical product under the reported test conditions.
[b] Technical product; experimental conditions (pH) were adjusted to increase solubility.
[c] 75% dry flowable formulation.

Source: DuPont, 1983b; Fred O'Neal, DuPont, Agricultural Products Department, Wilmington, Delaware, personal communication, 1987.

Table 6-18

Toxicity of tebuthiuron to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	144	96-hr LC_{50}	USDA, 1986
eggs and larvae	26	NOEL, no effects on hatchability, growth, behavior, development, or survival; reduced growth and survival at 52 ppm	USDA, 1986
Bluegill	112	96-hr LC_{50}	USDA, 1986
Fathead minnow	>160 (technical)	96-hr LC_{50}	Todd et al., 1974, as cited in USDA, 1986
eggs and larvae	9.3	NOEL, no effects on hatching, growth, development, behavior, or survival; reduced growth at 18 ppm	USDA, 1986
Goldfish	>160	96-hr LC_{50}	Todd et al., 1974, as cited in USDA, 1986
Fiddler crab	>320	96-hr LC_{50}, 320 ppm was highest concentration tested	USDA, 1986
Pink shrimp	48	96-hr LC_{50}	USDA, 1986
Water flea			
D. magna	297	48-hr EC_{50}	USDA, 1986
	21.8	No effects on reproduction, growth, or survival with lifetime exposure	USDA, 1986
Oyster			
embryos	180-320	48-hr EC_{50}, abnormal development	USDA, 1986

Table 6-19

Toxicity of triclopyr to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Rainbow trout	0.74[a]	96-hr LC_{50}	Dow Chemical Company, 1983, as cited in USDA, 1984
	552[b]	96-hr LC_{50}	Dow Chemical Company, 1983, as cited in USDA, 1984
	117	96-hr LC_{50}	Dow Chemical Company, 1983, as cited in USDA, 1984
Bluegill	0.87[a]	96-hr LC_{50}	Dow Chemical Company, 1983, as cited in USDA 1984
	891[b]	96-hr LC_{50}	
	148	96-hr LC_{50}	
Fathead minnow	120 (104-140)[b,c]	96-hr LC_{50} (Toxicity increased with temperature between 17 to 26 °C)	Mayes et al., 1984
	101 (88.5-116)[b,c]	8-day LC_{50}	Mayes et al., 1984
	245 (224-269)[b]	96-hr LC_{50} Static test	Mayes et al., 1984
embryo-larval stages	91[b]	MATC at 8 days based on mortality; no significant effects on hatchability, development, or growth	Mayes et al., 1984
Crab	>1,000[d]	96-hr LC_{50}	Dow Chemical Company, 1983, as cited in USDA, 1984

Table 6-19 (continued)

Toxicity of triclopyr to aquatic organisms

Species	Concentration (ppm)	Effect	Source
Shrimp	895[d]	96-hr LC_{50}	Dow Chemical Company, 1983, as cited in USDA, 1984
Water flea *D. magna*	1,170 (1,030-1,340)[b,e]	48-hr LC_{50}	Gersich et al., 1984
	1,140 (950-1,590)[b,e]	21-day LC_{50}	Gersich et al., 1984
	110[b]	MATC based on total young and brood size	Gersich et al., 1984
Oyster	56-87[d]	48-hr LC_{50}	Dow Chemical Company 1983, as cited in USDA, 1984

[a] Garlon 4 butoxyethyl ester.
[b] Garlon 3A triethylamine salt (TEA) or other TEA.
[c] Flow-through tests.
[d] Garlon 3A unspecified formulation.
[e] Range is 95% confidence interval.

Section 7

WILDLIFE AND AQUATIC SPECIES EXPOSURES

This section describes the estimated wildlife and aquatic species exposures to the 14 herbicides and additives used in Region 8. It discusses the representative species selected for exposure estimation and presents details of how exposures for each species were determined based on the species biology and the chemical application rates.

WILDLIFE EXPOSURES

Representative Wildlife Species

Wildlife exposures were calculated for a group of wildlife species representative of those typically found in areas supporting forest vegetation in the Southeast. These species represent a range of phylogenetic classes, body sizes, and diets. The methodology used to determine the exposures is the same as that used in the environmental impact statements prepared by the U.S. Department of Justice, Drug Enforcement Administration, on the eradication of cannabis with herbicides (U.S. Drug Enforcement Administration, 1985, 1986) and the environmental impact statement prepared by the U.S. Department of the Interior, Bureau of Land Management, on the control of noxious weeds with herbicides (Bureau of Land Management, 1987). Table 7-1 lists the representative wildlife species. Table 7-2 gives the various biological parameters used for each representative species in the exposure analysis.

Wildlife Data Sources

The references used in the species selection and in deriving the biological parameters of each species were the following:

(1) Distribution, Life History, and Diet
- Birds: Robbins et al. (1966), Scott et al. (1977), Chapman (1966), Meyers and Johnson (1978), Wood and Niles (1978), Dickson (1978), Beal (1911), U.S. EPA (1984), Prickett (undated).
- Mammals: Schmidt and Gilbert (1978), Burt and Grossenheider (1966), Hamilton and Whitaker (1979), Hamilton (1941), Sargeant (1978), Lockie (1959), Komarek and Komarek (1938), Odum (1949), Davis (1974), Davis (1978), Davis (1979), Lowery (1974).
- Reptiles and Amphibians: Conant (1958), Auffenberg and Iverson (1979), Seehorn (1982), Dickerson (1969).

(2) Physiology, Metabolism, Food Intake, and Weight
- Gordon et al. (1968), Hutchinson et al. (1968), Lasiewski and Dawson (1967), Kendeigh (1970), Lasiewski and Calder (1971), Schmidt-Nielsen (1975), Schmidt-Nielsen (1972), Sturkie (1965), Slobodkin (1961), Welty (1962), Zar (1968), Drozdz (1968), Odum (1971), Moore (1964), Altman and Dittmer (1962), U.S. EPA (1984), Kendeigh (1970), Seibert (1949), Banse and Mosher (1980), Odum et al. (1962), Damuth (1981), Kendeigh (1969).

Table 7-1

Representative southeastern wildlife species

Group	Common Name
Terrestrial vertebrates (class/food habit)	
Birds	
Insectivorous	Common flicker Red-cockaded woodpecker[a]
Granivorous	Bobwhite quail
Omnivorous	Eastern bluebird
Piscivorous	Belted kingfisher
Carnivorous	American kestrel
Mammals	
Insectivorous	Southern short-tailed shrew Red bat
Granivorous	Eastern gray squirrel
Small herbivorous	Pine vole
Medium herbivorous	Eastern cottontail
Large herbivorous	White-tailed deer Domestic cow
Small omnivorous	Cotton rat
Medium omnivorous	Eastern red fox
Large omnivorous	Black bear
Piscivorous	River otter
Carnivorous	Bobcat
Amphibians	
Insectivorous	Woodhouse toad

Table 7-1 (continued)

Representative southeastern wildlife species

Group	Common Name
Reptiles (food habit)	
Omnivorous	Eastern box turtle
Carnivorous	Hognose snake
Herbivorous	Gopher tortoise[b]
Invertebrates	Earthworm American bird grasshopper Leafcutting ant Honey bee

[a] Federally listed endangered species.
[b] Threatened in the western part of its range; a "sensitive" species in the eastern part of its range.

Wildlife Exposure Estimates

Realistic and extreme acute exposure estimates were made for each representative species for each of the three major exposure routes: inhalation, dermal, and ingestion. For several reasons--the herbicides degrade relatively rapidly, sites are normally treated only once in a given year, and operations are performed only 1 to 3 times per rotation or an average (in the most frequent case) of once in 20 years--no analysis of chronic wildlife dosing was done. Because the herbicides show no tendency to bioaccumulate, as discussed in section 3, long-term persistence in food chains and subsequent toxic effects were not considered a problem and were not examined in the risk analysis.

Herbicide doses for the representative species were calculated using conservative, simplified assumptions concerning routine application operations that give realistic dose estimates and highly unlikely (extreme) dose estimates in which animals are directly sprayed with herbicide. Exposures for realistic and extreme cases were based on the typical and maximum herbicide application rates for ground-mechanical applications (table 7-3).

For realistic doses, dermal exposures were based on the levels likely to be found on vegetation leaf surfaces because the animals are assumed to seek cover during a spraying operation. Extreme dose levels were estimated by assuming that animals do not seek cover and thus receive the full herbicide application rate on their entire body surface.

Table 7-2

Representative wildlife and domestic species
and associated biological parameters

Representative Niche	Representative Species	Body Weight (grams)	Daily Food Intake (grams)	Percentage of Food Contaminated in Realistic Case	Body Surface Area (cm^2)	Body Surface Contacting Vegetation (percent)	Percentage of Body Groomed	Inhalation Volume (1/min)
Insectivorous Birds	Common flicker	138	28	37	267	76	41	0.078
	Red-cockaded woodpecker[a]	50	12	46	136	84	55	0.003
Granivorous Bird	Bobwhite quail	170	34	36	307	74	39	0.092
Omnivorous Bird	Eastern bluebird	29	6	51	94	89	65	0.022
Piscivorous Bird	Kingfisher	250	60	33	398	71	35	0.125
Carnivorous Bird	American kestrel	112	26	39	233	78	44	0.066
Insectivorous Mammals	Southern short-tailed shrew	18	14	56	69	94	74	0.013
	Red bat	12	8	61	52	98	84	0.009
Granivorous Mammal	Eastern gray squirrel	425	45	30	566	67	30	0.147
Small herbivorous Mammal	Meadow vole	26	9	52	88	90	67	0.017
Medium herbivorous Mammal	Eastern cottontail	1,000	110	25	1,002	62	23	0.284
Large herbivorous Mammal	Deer	68,000	2,500	11	16,722	39	7	7.32
Small omnivorous Mammal	Cotton rat	156	45	36	290	75	40	0.068

Table 7-2 (continued)

Representative wildlife and domestic species and associated biological parameters

Representative Niche	Representative Species	Body Weight (grams)	Daily Food Intake (grams)	Percentage of Food Contaminated in Realistic Case	Body Surface Area (cm^2)	Body Surface Contacting Vegetation (percent)	Percentage of Body Groomed	Inhalation Volume (l/min)
Medium omnivorous Mammal	Eastern red fox	5,670	475	18	3,189	51	14	1.08
Large omnivorous Mammal	Black bear	92,000	4,470	10	20,457	38	6	9.23
Piscivorous Mammal	River otter	7,000	900	17	3,670	50	13	1.27
Carnivorous Mammal	Bobcat	6,000	520	18	3,311	51	14	1.13
Insectivorous Amphibian	Woodhouse toad	22	5	54	79	92	NA[b]	0.007
Omnivorous Reptile	Eastern box turtle	250	25	33	398	71	NA	0.003
Herbivorous Reptile	Gopher tortoise[c]	6,000	300	18	3,311	51	NA	0.003
Carnivorous Reptile	Hognose snake	40	22	48	117	86	NA	0.003
Domestic animals	Cattle	453,590	12,000	7	59,292	32	4	31.5
	Chicken	2,000	300	22	1,591	57	19	0.66
	Dog	13,000	NA	NA	5,546	47	11	2.95

[a] Federally listed endangered species.
[b] NA = Not applicable or not available.
[c] USDA Forest Service species of concern.

Table 7-3

Typical and maximum rates for ground-mechanical applications in Region 8

Herbicide/Additive	Typical Rate kg/ha	Typical Rate (lb/ac)	Maximum Rate kg/ha	Maximum Rate (lb/ac)
2,4-D (ester)	4.48	(4.0)	7.85	(7.0)
2,4-DP	4.48	(4.0)	6.73	(6.0)
Dicamba	2.24	(2.0)	3.36	(3.0)
Diesel oil	2.24	(2.0)	3.92	(3.5)
Fosamine	8.69	(7.75)	13.45	(12.0)
Glyphosate	1.68	(1.5)	4.48	(4.0)
Hexazinone	1.91	(1.7)	6.73	(6.0)
Imazapyr	0.84	(0.75)	1.68	(1.5)
Kerosene	2.54	(2.27)	5.09	(4.54)
Limonene	1.00	(0.9)	4.04	(3.6)
Picloram	0.78	(0.7)	1.57	(1.4)
Sulfometuron methyl	0.19	(0.17)	0.41	(0.37)
Tebuthiuron	1.12	(1.0)	6.73	(6.0)
Triclopyr (ester)	4.48	(4.0)	8.97	(8.0)

The dermal penetration rates used in the human exposure analysis were used to determine mammalian wildlife dermal penetration (that is, the amount of chemical that penetrates the animal's skin). A dermal penetration rate of 10 percent was assumed for the herbicides for which no dermal penetration data were available. In both realistic and extreme exposures, mammals and birds are assumed to receive an oral dose from grooming their fur or preening their feathers. This amount is subtracted from the amount they would receive from their dermal exposure.

Because larger animals have larger home ranges, they are not as likely to feed on contaminated items at a particular site as are smaller animals. Therefore, realistic ingestion doses were assumed to come from animals eating a specified percentage of their daily food intake in contaminated items based on their body size. That is, the percentage of contaminated food intake decreases as body size increases. In the extreme case, the animals are assumed to feed entirely on contaminated food items.

Inhalation exposures are assumed to come from a hypothetical amount of herbicide droplets forming a "cloud" that moves slowly offsite.

The total systemic dose to each animal was calculated as the sum of the estimated doses received via dermal, ingestion, and inhalation routes. Tables 8-1 to 8-14 in the wildlife risk analysis section (section 8) give the total realistic and extreme dose estimates for the representative species.

Exposure Calculations

Inhalation Exposures. Wildlife inhalation exposures were assumed to come from animals breathing in herbicide spray droplets of respirable size (30 microns in diameter or less) as a hypothetical "cloud" of those droplets moves slowly offsite. The cloud is assumed to be dispersed within the first 5 m above ground level on a 16.2-ha (40 ac) site 402 m on a side and to consist of respirable droplets that constitute 1 percent of the total applied herbicide by volume. Based on these assumptions, the airborne concentration is 0.0002242 mg/l for each 1.12 kg/ha (1 lb/ac) applied. The cloud moves offsite at 0.9 m/sec (2 mph) and exposes animals on the downwind edge for 7.5 minutes in the realistic case. The wind is assumed to be 0.45 m/sec (1 mph) in the extreme case so that animals are exposed for 15 minutes. The nominal exposure was multiplied by the herbicide application rate and then by each animal's breathing rate. Their breathing rate in liters per minute is based on the following equations:

Birds: $$LPM = \frac{284 \times (BWT/1000)^{.77}}{1000}$$

Mammals: $$LPM = \frac{379 \times (BWT/1000)^{.80}}{1000}$$

Reptiles: $LPM = .00334$

Amphibians: $LPM = .007$

where:

LPM is the animal's breathing rate in liters per minute

BWT is the animal's body weight in grams

The equations for birds and mammals were taken from Lasiewski and Calder (1971). The reptile value is from Gordon et al. (1968), who report a study on the collared lizard. The breathing rate for amphibians was from Hutchinson et al. (1968). As anticipated, the animal modeling results showed inhalation exposures to be only a small fraction of each species total dose.

Dermal Exposures. Dermal exposures are assumed to come from two sources: (1) directly from herbicide spray at the deposition rate that should occur on vegetation leaf surfaces in the realistic case and at the herbicide application rate in the extreme case, and (2) indirectly by contact with contaminated vegetation.

Fur, feathers, and scales afford varying degrees of protection against dermal exposure; by preventing the chemical from reaching the animal's skin, they may instead allow the chemical to dry or to be rubbed off in their movements. For this reason, the dermal penetration rate for each herbicide for mammals was adjusted for three other animal classes--birds, reptiles, and amphibians. Dermal penetration factors were multiplied by the mammalian penetration rate as follows: (1) birds, 0.75; (2) reptiles, 0.15; and (3) amphibians, 5.0. The amphibian factor is high because the moist, glandular skin of the amphibian serves to a large extent as a respiratory organ and is much more permeable than the skin of the other animal classes (30 percent (5 to 93 percent) of body weight in water moves through skin in 24 hours according to Moore, 1964).

Wildlife may receive indirect dermal exposure from moving through contaminated vegetation by transferring pesticide from the vegetation to their body surface. The amount transferred would depend on (1) the density of the vegetation, (2) the animal's body size in relation to the height of the vegetation, and (3) the amount of movement of the animal.

To simplify the analysis, it was assumed that a certain percentage of the animal's total body surface received herbicide at the same level as the direct dermal exposure (either the level on leaf surfaces in the realistic case or at the application rate in the extreme case). That percentage was based on the animal's body size and a movement factor (MVF) to adjust for the taxonomic class. (Mammals, for example, are expected to move more than amphibians.) The animal's total body surface area was assumed to be a function of its weight according to the following formula (Kendeigh, 1970; Schmidt-Nielsen, 1972):

$$BSA = 10 \times (BWT)^{.667}$$

where:

BSA is the animal's body surface area in cm^2

BWT is the animal's body weight in grams

The animal's vegetation contact percent (VCP) is based on its body weight in grams (BWT) according to the following formula:

$$VCP = 2.89 \ (BWT)^{-.3775}$$

The class adjustment factors (MVF's) for differing movement are as follows: (1) birds, 0.8; (2) mammals, 1; (3) reptiles, 0.3; and (4) amphibians, 0.4. The indirect dermal dose (IND) is then calculated using the direct dermal dose (DDD):

$$IND = DDD + (DDD \times VCP \times MVF)$$

Mammals and birds groom themselves regularly and may receive an ingestion dose if their fur or feathers are contaminated. The percent of their body surface groomed (PBG) was assumed to be a decreasing function of their body size according to the following formula:

$$PBG = 1.72 \ (BWT)^{-.29}$$

No grooming was assumed for reptiles and amphibians. The oral dose for mammals and birds from grooming was subtracted from the amount of herbicide that would contribute to the animal's dermal dose.

Ingestion Doses. Each representative species was assumed to feed on contaminated food items according to a specified diet and to drink a specified amount of water. These dietary amounts are listed in table 7-4. Diets may vary from season to season and across the species range; the diet items and amounts were chosen to be a reasonable representation of what an individual animal might consume on a given day. The diet items--grass, forage vegetation, seeds, insects, and berries--are assumed to have the following contamination levels in ppm from ground application based on field studies by Hoerger and Kenaga (1972) for a 1-lb/ac application rate:

	Realistic	Extreme
	ppm	
Grass	1.665	92
Forage	0.439	33
Seeds	0.040	3.2
Insects	0.0627	4.8
Berries	0.0199	1.6

Water is assumed to be drunk in the realistic case from a stream offsite that reaches a concentration of 0.001267 ppm per pound of herbicide applied per acre for aerially applied herbicides and 0.0003 ppm for ground-applied herbicides. In the extreme case, water reaches a concentration of 0.0068 ppm for aerially applied herbicides and 0.00063 ppm for ground-applied herbicides. Predators that feed on mice or toads are assumed to receive the total body burden that each of these prey species has received through the three exposure routes described above as a result of the herbicide spraying operation. Predators that feed on fish (piscivores) are assumed to receive residue levels based on the concentration in the water. In the realistic exposures, each species is assumed to consume a percentage of its daily intake in contaminated food items depending on its body size. The percentages of food contaminated (PFC) (listed in table 7-2) are based on the following formula:

$$PFC = 100 \times (1/(BWT)^{.2}$$

In the extreme case, each species' entire daily food intake is assumed to consist of herbicide-contaminated items.

AQUATIC SPECIES EXPOSURES

Representative Aquatic Species

Representative species typical of aquatic habitats in the Southeast are given in table 7-5. These species were assumed to be exposed by immersion to estimated concentrations of the 11 herbicides and 3 additives in bodies of water with specified characteristics.

Table 7-4

Representative wildlife species diet items[a]

Representative Species	Water	Grass	Forage	Seeds	Insects	Berries	Mouse	Toad	Fish
Birds									
Flicker	0.02	0	0	0	28	0	0	0	0
Bobwhite quail	0.10	0	0	26	4	4	0	0	0
Eastern bluebird	0.018	0	0	1	3	2	0	0	0
Belted kingfisher	0.075	0	0	0	0	0	0	0	60
American kestrel	0.05	0	0	0	0	0	26	0	0
Red-cockaded woodpecker[b]	0.04	0	0	0	12	0	0	0	0
Mammals									
Southern short-tailed shrew	0.02	0	0	0	14	0	0	0	0
Red bat	0.015	0	0	0	8	0	0	0	0
Eastern gray squirrel	0.15	0	0	45	0	0	0	0	0
Meadow vole	0.03	8	0	1	0	0	0	0	0
Eastern cottontail	0.25	110	0	0	0	0	0	0	0
White-tailed deer	1.5	500	2,000	0	0	0	0	0	0
Cotton rat	0.15	45	0	0	0	0	0	0	0
Eastern red fox	0.5	0	0	0	0	175	300	0	0
Black bear	1.8	200	400	450	700	1,200	520	0	1,000
River otter	0.5	0	0	0	0	0	0	0	900
Bobcat	0.4	0	0	0	0	0	520	0	0
Amphibian									
Woodhouse toad	0.02	0	0	0	5	0	0	0	0
Reptile									
Eastern box turtle	0.07	0	0	0	25	0	0	0	0
Hognose snake	0.02	0	0	0	0	0	0	22	0
Gopher tortoise[c]	0.35	150	150	0	0	0	0	0	0
Domestic animals									
Cattle	58	12,000	0	0	0	0	0	0	0
Chicken	0.07	0	0	300	0	0	0	0	0
Dog	0.50	0	0	0	0	0	0	0	0

[a] Consumption is in liters for water and in grams for all other items.

[b] Federally listed endangered species.

[c] USDA Forest Service species of concern.

Table 7-5

Representative aquatic species

Class/Food Habit	Family	Common Name
Fish		
Insectivorous-piscivorous	Salmonidae	Rainbow trout
Insectivorous		Brook trout
Insectivorous-piscivorous	Centrarchidae	Largemouth bass
Insectivorous-piscivorous		Smallmouth bass
Insectivorous		Bluegill
Insectivorous-piscivorous		Green sunfish
Omnivorous	Cyprinidae	Fathead minnow
Herbivorous	Clupeidae	Gizzard shad
Omnivorous	Catostomidae	Northern hogsucker
Insectivorous	Poeciliidae	Mosquitofish
Piscivorous	Esocidae	Chain pickerel
Invertebrates		
Herbivorous		Crayfish
Detritivorous		Water flea
Herbivorous-omnivorous		Stonefly nymph
Detritivorous		Eastern or Virginia oyster
Amphibia		
Insectivorous	Necturidae	Mudpuppy

Aquatic Exposure Estimates

Exposure was assumed to occur for herbicides that drift offsite from mechanical ground applications. Typical and maximum estimated environmental concentrations (EEC's) of each herbicide were computed for a body of water 0.61 m (2 ft) deep (see table 7-6) as described in the human exposure analysis in section 3. Typical EEC's were based on typical application rates and a distance of 20.1 m (66 ft) from the application site to the body of water; maximum EEC's were calculated using maximum application rates and a distance of 10.1 m (33 ft) to a water body. EEC's for kerosene were based on the fraction of kerosene in triclopyr ester formulations.

To assess the effects of accidents, aquatic EEC's were calculated for a spill of an 18.9-liter (5-gal) can of herbicide into a pond and a spill of a 378.5-liter (100-gal) helicopter load of herbicide mixture into a reservoir (table 7-6). In all cases, the spill into the pond results in higher EEC's than the spill into the reservoir. Concentrations were also calculated for accidental direct spraying of a body of water (table 7-6). The exposure levels from the typical and maximum EEC's and from the accident EEC's are described in section 8 on the aquatic species risk analysis.

Table 7-6

Herbicide concentrations in water
(ppm)

Herbicide	Offsite Drift		Spill in Pond	Spill in Reservoir	Direct Spraying	
	Typical	Maximum			Typical	Maximum
2,4-D amine	0.0016	0.0036	1.7	--[a]	0.736	1.288
2,4-D ester	0.0025	0.0063	1.7	--[a]	0.736	1.288
2,4-DP	0.0025	0.0054	1.7	--[a]	0.736	1.104
Dicamba	0.0013	0.0027	0.46	--[a]	0.368	0.552
Diesel fuel	0.0013	0.0031	3.1	0.043	0.368	0.644
Fosamine	0.0049	0.011	1.8	--[a]	1.426	2.208
Glyphosate (Rodeo)	0.0010	0.0036	1.4	0.09	0.276	0.736
Glyphosate (Roundup)	0.0010	0.0036	1.4	0.09	0.276	0.736
Hexazinone	0.0011	0.0054	0.92	--[a]	0.3126	1.104
Imazapyr	0.00048	0.0013	0.92	0.043	0.138	0.276
Kerosene	0.0014	0.0041	1.0	0.13	0.41768	0.83536
Limonene	0.00057	0.0032	3.3	0.052	0.1656	0.6624
Picloram and 2,4-D	0.00044	0.0013	0.12	--[a]	0.1288	0.2576
Sulfometuron methyl	0.00011	0.00033	1.6	--[a]	0.03128	0.06808
Tebuthiuron	0.00020	0.0023	3.7	0.17	0.184	1.104
Triclopyr amine	0.0025	0.0072	1.4	--[a]	0.736	1.472
Triclopyr ester	0.0025	0.0072	1.8	0.23	0.736	1.472

[a] No aerial use.

Section 8

WILDLIFE AND AQUATIC SPECIES RISK ANALYSIS

The risk analysis considers potential wildlife and aquatic species impacts of using 14 herbicides and additives in the Region 8 vegetation management program. It determines that, even using very conservative assumptions to estimate possible exposures, in general, risks to wildlife and aquatic species from the Forest Service's vegetation management program are low.

Wildlife and aquatic species risk from vegetation management with herbicides is a function of the inherent toxicity (hazard) of each herbicide to different organisms and of the amount of each chemical (exposure) those organisms may take in as a result of a vegetation management operation. As in the analysis of human health effects, the wildlife and aquatic species risk analysis compares estimated acute exposures of representative species determined in the previous section with acute toxicity levels found in laboratory studies. Common and scientific names for all of the representative species are listed in table 8-35 at the end of this section.

WILDLIFE RISK ANALYSIS

Wildlife Risk Analysis Criteria

For wildlife risks, the criteria used by EPA in ecological risk assessment (EPA, 1986) were used to judge the absolute risks to the different representative species and the relative risks among the 14 herbicides and additives. The EPA criteria call for comparison of an estimated environmental concentration (EEC) with a laboratory-determined LD_{50} or LC_{50} for the most closely related laboratory test species.

Where the EEC exceeds 1/5 LD_{50} or LC_{50}, EPA deems it a significant risk that may be mitigated by restricting use of the pesticide. EPA judges EEC's that exceed the LD_{50} or LC_{50} as unacceptable risk levels. Doses below the 1/5 LD_{50} level are assumed to present a low risk. In this risk assessment, an organism's total estimated dose (rather than an EEC) is compared with the laboratory toxicity level because the dose comes from all exposure routes, not just feeding.

Wildlife Toxicity Surrogates

There is a considerable amount of uncertainty in the toxicity data and methods used in the wildlife risk assessment.

The toxicity of herbicides to wildlife varies among individuals of the same species (intraspecific), between different species (interspecific), and, often most markedly, between different classes of animals. Thus, an herbicide may be more toxic to birds than to mammals, or more toxic to fish than to birds. However, toxicity testing has been conducted on relatively few wildlife species, and the testing has been confined to a few avian and mammalian wildlife species. Laboratory animal studies have been done on inbred strains of test animals, particularly rats and mice, to estimate human toxicity.

An analysis of the herbicide risk to wildlife compared estimated acute doses for the representative wildlife species described in section 7 with available hazard information on the most closely related species as described in section 6. Because the herbicides examined in this appendix show no tendency to bioaccumulate, long-term persistence in food chains and subsequent toxic effects, such as those that have resulted from the use of the persistent organochlorides, are not considered a problem and are not examined in the risk analysis. No analysis of chronic wildlife dosing was done for several reasons--the herbicides degrade relatively rapidly, sites are normally treated only once in a given year, and applications on a given site are performed only 1 to 3 times per rotation, or an average (in the most frequent case) of once in 20 years.

Surrogates for Avian and Mammalian Toxicity

Toxicity data on the most closely related avian or mammalian species are used for the wildlife risk comparisons. Except for limonene, herbicides and additives have been tested on at least one bird species. Mallard data are used only when no data on an upland species, such as the bobwhite, japanese quail, or pheasant, are available. Where no data on a mammalian wildlife species (for example, mule deer) are available, data on laboratory rats, mice, dogs, rabbits, or guinea pigs are used for comparison with representative species doses.

Surrogates for Amphibian and Reptile Toxicity

The U.S. Fish and Wildlife Service, in its testing of nearly 200 chemicals on terrestrial vertebrate wildlife species (Hudson et al., 1984), tested 19 pesticides, principally organophosphate and carbamate insecticides, on the adult stage of the bullfrog. No tests were done on reptiles, and none of the herbicides and additives being evaluated for Region 8 were used in the tests on the bullfrog. There was a good correlation ($r = 0.67$) between the LD_{50}'s for the bullfrog and the LD_{50}'s for the mallard for the tested chemicals when 17 of the 19 chemicals were used in a prediction equation. The bullfrog LD_{50}'s for 14 of the 19 pesticides were higher than those of the mallard.

In its studies of aquatic species (Mayer and Ellerseick, 1986), the U.S. Fish and Wildlife Service tested 20 and 13 pesticides, respectively, on the immature stage (tadpole) of two amphibian species--Fowler's toad and the western chorus frog. Most of the tests were on organochloride and organophosphate insecticides. One of the herbicides being evaluated for Region 8, 2,4-D butoxyethanol ester, was tested on the Fowler's toad. There was a poor correlation (r less than 0.10) between the tadpole LC_{50}'s and mallard or rat LD_{50}'s for the same pesticides. Johnson (1976) reported studies of herbicide toxicity on 1- to 2-week-old tadpoles of three species of Australian amphibians. Picloram, 2,4-D, and dicamba were among the 10 herbicides tested. In neither study were the data amenable to a translation from LC_{50}'s for the tadpoles (from immersion exposure) to LD_{50}'s for the adult stage for exposure from dermal, ingestion, and inhalation.

The U.S. Fish and Wildlife Service also reviewed the available data on the toxicity of environmental contaminants to reptiles (Hall, 1980). Most of the data consisted of residue levels of organochlorides in reptiles collected after field applications. There were no data of the type reported in the above amphibian studies relating dose levels to lethality; however, the author noted that bird data could serve as a guide for reptile toxicity because birds were closely related to reptiles, although, in general, reptiles appeared to be more susceptible to pesticides than birds or mammals.

Thus, for the 14 herbicides and additives in this risk assessment, suitable data are lacking for terrestrial stages of amphibians and for reptiles. Because there is a reasonable correlation between avian and amphibian toxicity as indicated in the mallard versus bullfrog LD_{50} analysis and reason to suspect the same of avian and reptilian toxicity as noted by Hall (1980), available avian toxicity data were used as surrogates for both amphibians and reptiles.

Wildlife toxicity reference levels used to assess the risks of the 11 herbicides and 3 related additives are given in tables 8-1 through 8-14.

Wildlife Exposure Analysis

Tables 8-1 through 8-14 give the total realistic and extreme dose estimates for the 24 representative wildlife species for each of the herbicides and additives being evaluated for Region 8.

The wildlife risk assessment tends to overstate the risks because many of the assumptions are quite conservative. For example, no degradation of the herbicides is assumed to occur and all herbicide sprayed is assumed to be biologically available. In the extreme exposures, the entire diet of an animal is assumed to consist of contaminated items, while in the realistic case, a significant percentage (7 to 61 percent, depending on body size based on exposure modeling assumptions) of the diet is assumed to be contaminated. Dermal exposures are assumed to come both directly from herbicide spray and indirectly from brushing up against treated vegetation. Birds and mammals are assumed to receive dermal doses through their skin and from grooming. This accumulation of doses from almost every conceivable route undoubtedly overestimates doses, even in the realistic case. Nevertheless, when these dose estimates do exceed the EPA risk criterion, and more so when they exceed the LD_{50} for the most closely related laboratory species, there is a clear risk of adverse effects on individual animals.

Wildlife Risk Overview

In general, based on the available toxicity data and on the proposed application rates, the risks to wildlife from the use of the 11 herbicides and 3 additives are low to negligible in the Region 8 vegetation management program. Estimated doses for realistic exposures exceed 100 mg/kg only for one herbicide, fosamine, and then only in one species, the red bat. Except for small mammals and the smaller birds, realistic doses seldom exceed 10

best copy available

mg/kg for any of the herbicides. The realistic dose estimates are below the EPA risk criterion of 1/5 LD_{50} and are far below the laboratory species LD_{50} for the majority of the chemicals.

2,4-D and 2,4-DP present the highest relative risks to wildlife of the herbicides considered, although their absolute risks are moderate. Hexazinone, tebuthiuron, and triclopyr present low to moderate risks to wildlife. Glyphosate presents a low to very low wildlife risk. Fosamine, imazypyr, kerosene, limonene, picloram, and sulfometuron methyl present the lowest wildlife risks.

Local populations of small mammals, small birds, terrestrial amphibians, and reptiles may be adversely affected if large areas are treated; however, the reproductive capacity of these species is generally high enough to replace the few lost individuals within the next breeding cycle. Populations of larger mammals and birds and any domestic animals present are not likely to be affected at all.

The risks of the individual herbicides are discussed below. Literature references for the toxicity levels in laboratory species are given in the wildlife hazard analysis. Again, it must be noted that there are very few toxicity studies on which to base these conclusions. Avian toxicity data are particularly rare for most of the herbicides. 2,4-DP, glyphosate, hexazinone, kerosene, and sulfometuron methyl had only two or three laboratory animal LD_{50} tests to use in the analysis. Limonene had only a single rat oral LD_{50} to use. However, the conservatism used in estimating the wildlife doses should compensate for much of the uncertainty in the toxicity data base.

Wildlife Risk From the Individual Herbicides

The risks to wildlife from the use of 2,4-D are moderate. Estimated realistic wildlife doses (table 8-1) range from 12 to 35 mg/kg for birds and from less than 1 mg/kg for larger mammals to 62 mg/kg for small mammals. These doses are below the EPA 1/5 LD_{50} criterion for avian species. Small mammal doses approach the EPA level. Realistic doses for larger mammals, amphibians, and reptiles are well below the EPA level. Extreme dose levels for the majority of representative species approach or exceed the EPA risk level. Extreme doses for the bluebird, shrew, red bat, and vole exceed the laboratory LD_{50}.

Estimated wildlife dose levels of 2,4-DP (table 8-2) are comparable to those of 2,4-D. Small mammals may be at a moderate level of risk from the use of 2,4-DP. Their realistic dose levels represent a significant portion of the EPA 1/5 LD_{50} risk level. Birds may also be at moderate risk. However, it must be noted that the LD_{50} for avian species is based on the lethal level for 2,4-D in the Japanese quail and chukar because avian data on 2,4-DP are lacking. The extreme estimated doses for birds and small mammals exceed the EPA levels. It does not appear that larger mammals, amphibians, or reptiles are at risk from 2,4-DP use.

Dicamba realistic doses (table 8-3) are well below the EPA 1/5 LD_{50} risk level for all representative species. Small mammal and smaller bird extreme doses approach the 1/5 LD_{50} level. The red bat and meadow vole

Table 8-1

2,4-D wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	16	143	60	300	Chukar
Bobwhite quail	14	127	134	668	Japanese quail
Eastern bluebird	35	309	60	300	Chukar
Belted kingfisher	12	101	60	300	Chukar
American kestrel	27	282	60	300	Chukar
Red-cockaded woodpecker[a]	26	238	60	300	Chukar
Mammals					
So. s-tail shrew	50	461	76	380	Mouse
Red bat	62	560	76	380	Mouse
E. gray squirrel	10	89	75	375	Rat
Meadow vole	42	559	76	380	Mouse
E. cottontail	7	127	84	424	Rabbit
White-tailed deer	0.9	19	120	600	Deer
Cotton rat	17	329	75	375	Rat
Eastern red fox	5	54	20	100	Dog
Black bear	1	13	20	100	Dog
River otter	2	22	20	100	Dog
Bobcat	6	72	20	100	Dog
Amphibians					
Woodhouse toad	22	199	60	300	Chukar
Reptiles					
E. box turtle	9	79	60	300	Chukar
Hognose snake	29	254	60	300	Chukar
Gopher tortoise[b]	0.5	26	60	300	Chukar
Domestic animals					
Cow	0.4	20	10	50	Cow
Chicken	4	39	76	380	Chicken
Dog	2	16	20	100	Dog

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-2

2,4-DP wildlife and domestic animal doses
compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	----------	----------	(mg/kg)	----------	----------
Birds					
Common flicker	15	115	60	300[a]	Chukar
Bobwhite quail	13	102	134	668[a]	Japanese quail
Eastern bluebird	34	256	60	300	Chukar
Belted kingfisher	11	80	60	300	Chukar
American kestrel	26	231	60	300	Chukar
Red-cockaded woodpecker[b]	25	195	60	300	Chukar
Mammals					
So. s-tail shrew	48	382	130	650	Mouse
Red bat	60	476	130	650	Mouse
E. gray squirrel	9	68	106	532	Rat
Meadow vole	40	465	130	650	Mouse
E. cottontail	6	103	106	532	Rat
White-tailed deer	0.6	14	106	532	Rat
Cotton rat	16	272	106	532	Rat
Eastern red fox	4	42	106	532	Rat
Black bear	0.8	9	106	532	Rat
River otter	2	15	106	532	Rat
Bobcat	6	57	106	532	Rat
Amphibians					
Woodhouse toad	0.3	8	60	300	Chukar
Reptiles					
E. box turtle	0.1	3	60	300	Chukar
Hognose snake	0.3	6	60	300	Chukar
Gopher tortoise[c]	0.04	19	60	300	Chukar
Domestic animals					
Cow	0.3	16	106	532	Rat
Chicken	4	30	60	300	Chukar
Dog	1	11	106	532	Rat

[a] Based on the 2,4-D LD_{50}.
[b] Federally listed endangered species.
[c] Federally listed threatened species.

Table 8-3

Dicamba wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	9	61	135	673	Pheasant
Bobwhite quail	7	54	135	673	Pheasant
Eastern bluebird	17	132	135	673	Pheasant
Belted kingfisher	6	43	135	673	Pheasant
American kestrel	14	121	135	673	Pheasant
Red-cockaded woodpecker[a]	13	102	135	673	Pheasant
Mammals					
So. s-tail shrew	25	198	238	1189	Mouse
Red bat	31	240	238	1189	Mouse
E. gray squirrel	5	38	151	757	Rat
Meadow vole	21	240	238	1189	Mouse
E. cottontail	3	55	400	2000	Rabbit
White-tailed deer	0.4	8	400	2000	Rabbit
Cotton rat	9	141	151	757	Rat
Eastern red fox	2	23	151	757	Rat
Black bear	0.5	5	151	757	Rat
River otter	1	9	151	757	Rat
Bobcat	3	31	151	757	Rat
Amphibians					
Woodhouse toad	11	85	135	673	Pheasant
Reptiles					
E. box turtle	4	34	135	673	Pheasant
Hognose snake	14	109	135	673	Pheasant
Gopher tortoise[b]	0.2	11	135	673	Pheasant
Domestic animals					
Cow	0.2	9	400	2000	Rabbit
Chicken	2	17	135	673	Pheasant
Dog	0.9	7	151	757	Rat

[a] Federally listed endangered species.
[b] Federally listed threatened species.

doses exceed it. So dicamba presents a lower risk to wildlife than either 2,4-D or 2,4-DP, although a few animals could be seriously affected or killed.

Diesel oil realistic and extreme doses (table 8-4) are all well below the EPA risk level. No species should be directly affected in Region 8 by the use of diesel oil.

Fosamine presents a negligible risk of wildlife effects even though the estimated wildlife dose levels (table 8-5) are higher than those from 2,4-D or 2,4-DP use because the fosamine laboratory animal LD_{50}'s range from 5,000 to 24,400 mg/kg. As was the case with diesel oil, the realistic and extreme estimated doses of fosamine are well below the EPA 1/5 LD_{50} risk levels.

The analysis indicates that estimated wildlife doses of glyphosate (table 8-6), pose a very low risk to wildlife from both realistic and extreme exposures. Only small mammals could be considered at any degree of risk because their extreme doses are a significant fraction of the EPA 1/5 LD_{50} level. Birds and larger mammals, reptiles, and amphibians appear to be at very low to negligible risk from glyphosate.

Hexazinone presents a low to moderate degree of risk to wildlife. The extreme doses to small mammals exceed the EPA risk level (table 8-7). The extreme doses to birds, amphibians, and reptiles represent significant portions of the EPA risk level, although none approaches the LD_{50}. Hexazinone risks to larger mammals appears to be negligible.

Imazapyr risks to wildlife are low to negligible based on the limited amount of laboratory data available (table 8-8). The highest estimated doses are the extreme doses to small mammals that range up to 120 mg/kg. The lowest EPA risk level is 400 mg/kg. No animals should die from imazapyr exposures, and there should be few if any sublethal effects.

Kerosene, limonene, picloram, and sulfometuron methyl (tables 8-9 to 8-12) also present extremely low risks to wildlife again based on the very limited data available. Tebuthiuron (table 8-13) presents a very low risk to all wildlife species in the realistic exposure situations and a low risk to all wildlife species except small mammals under the extreme case exposures. Tebuthiuron wildlife risk appears to be lower than 2,4-D or 2,4-DP but higher than dicamba.

Triclopyr estimated doses (table 8-14), comparable to the doses of 2,4-D in the realistic case and slightly higher in the extreme case, present low to moderate risks to wildlife. Realistic doses are all below the EPA 1/5 LD_{50} risk levels but extreme doses exceed the EPA levels in one bird and several mammals. Small mammal extreme doses approach the laboratory animal LD_{50}'s and exceed them in the cases of the shrew, red bat, and meadow vole.

Table 8-4

Diesel oil wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	9	78	3280	16400	Mallard
Bobwhite quail	8	70	3280	16400	Mallard
Eastern bluebird	18	163	3280	16400	Mallard
Belted kingfisher	6	56	3280	16400	Mallard
American kestrel	15	150	3280	16400	Mallard
Red-cockaded woodpecker[a]	14	127	3280	16400	Mallard
Mammals					
So. s-tail shrew	26	243	1476	7380	Rat
Red bat	32	292	1476	7380	Rat
E. gray squirrel	6	52	1476	7380	Rat
Meadow vole	22	291	1476	7380	Rat
E. cottontail	4	70	1476	7380	Rat
White-tailed deer	0.6	11	1476	7380	Rat
Cotton rat	10	174	1476	7380	Rat
Eastern red fox	3	31	1476	7380	Rat
Black bear	0.7	8	1476	7380	Rat
River otter	2	14	1476	7380	Rat
Bobcat	4	40	1476	7380	Rat
Amphibians					
Woodhouse toad	27	243	3280	16400	Mallard
Reptiles					
E. box turtle	11	96	3280	16400	Mallard
Hognose snake	36	314	3280	16400	Mallard
Gopher tortoise[b]	0.6	16	3280	16400	Mallard
Domestic animals					
Cow	0.3	11	1476	7380	Rat
Chicken	2	23	3280	16400	Mallard
Dog	1	11	1476	7380	Rat

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-5

Fosamine wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	----------	---------- (mg/kg)	----------	----------	----------
Birds					
Common flicker	30	246	1000	5000	Bobwhite
Bobwhite quail	27	218	1000	5000	Bobwhite
Eastern bluebird	67	530	1000	5000	Bobwhite
Belted kingfisher	22	173	1000	5000	Bobwhite
American kestrel	53	483	1000	5000	Bobwhite
Red-cockaded woodpecker[a]	51	407	1000	5000	Bobwhite
Mammals					
So[illegible]s-tail shrew	97	790	4880	24400	Rat
Red bat	119	960	4880	24400	Rat
E. gray squirrel	19	152	4880	24400	Rat
Meadow vole	82	958	4880	24400	Rat
E. cottontail	13	218	1476	7380	Guinea pig
White-tailed deer	2	33	1476	7380	Guinea pig
Cotton rat	33	565	4880	24400	Rat
Eastern red fox	10	93	3000	15000	Dog
Black bear	2	22	3000	15000	Dog
River otter	5	38	3000	15000	Dog
Bobcat	12	123	3000	15000	Dog
Amphibians					
Woodhouse toad	42	341	1000	5000	Bobwhite
Reptiles					
E. box turtle	17	135	1000	5000	Bobwhite
Hognose snake	55	436	1000	5000	Bobwhite
Gopher tortoise[b]	0.9	44	1000	5000	Bobwhite
Domestic animals					
Cow	0.7	35	1476	7380	Guinea pig
Chicken	8	67	1000	5000	Bobwhite
Dog	4	28	3000	15000	Dog

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-6

Glyphosate wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	12	123	928	4640	Quail
Bobwhite quail	10	109	928	4640	Quail
Eastern bluebird	26	269	928	4640	Quail
Belted kingfisher	9	87	928	4640	Quail
American kestrel	20	241	928	4640	Quail
Red-cockaded woodpecker[a]	20	204	928	4640	Quail
Mammals					
So. s-tail shrew	37	395	800	4000	Rat
Red bat	46	480	800	4000	Rat
E. gray squirrel	7	76	800	4000	Rat
Meadow vole	32	479	800	4000	Rat
E. cottontail	5	109	760	3800	Rabbit
White-tailed deer	0.7	16	760	3800	Rabbit
Cotton rat	13	282	800	4000	Rat
Eastern red fox	4	46	800	4000	Rat
Black bear	0.7	11	800	4000	Rat
River otter	2	19	800	4000	Rat
Bobcat	5	62	800	4000	Rat
Amphibians					
Woodhouse toad	16	171	928	4640	Quail
Reptiles					
E. box turtle	6	68	928	4640	Quail
Hognose snake	21	218	928	4640	Quail
Gopher tortoise[b]	0.3	22	928	4640	Quail
Domestic animals					
Cow	0.3	17	760	3800	Rabbit
Chicken	3	33	928	4640	Quail
Dog	1	14	800	4000	Rat

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-7

Hexazinone wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	7	123	452	2258	Bobwhite
Bobwhite quail	6	109	452	2258	Bobwhite
Eastern bluebird	15	265	452	2258	Bobwhite
Belted kingfisher	5	87	452	2258	Bobwhite
American kestrel	12	241	452	2258	Bobwhite
Red-cockaded woodpecker[a]	11	204	452	2258	Bobwhite
Mammals					
So. s-tail shrew	21	395	338	1690	Rat
Red bat	26	480	338	1690	Rat
E. gray squirrel	4	76	338	1690	Rat
Meadow vole	18	479	338	1690	Rat
E. cottontail	3	109	172	860	Guinea pig
White-tailed deer	0.4	16	172	860	Guinea pig
Cotton rat	7	282	338	1690	Rat
Eastern red fox	2	46	338	1690	Rat
Black bear	0.4	11	338	1690	Rat
River otter	1	19	338	1690	Rat
Bobcat	3	62	338	1690	Rat
Amphibians					
Woodhouse toad	9	171	452	2258	Bobwhite
Reptiles					
E. box turtle	4	68	452	2258	Bobwhite
Hognose snake	12	218	452	2258	Bobwhite
Gopher tortoise[b]	0.2	22	452	2258	Bobwhite
Domestic animals					
Cow	0.2	17	172	860	Guinea pig
Chicken	2	33	452	2258	Bobwhite
Dog	0.8	14	338	1690	Rat

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-8

Imazapyr wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	3	31	430	2150	Bobwhite
Bobwhite quail	3	27	430	2150	Bobwhite
Eastern bluebird	7	66	430	2150	Bobwhite
Belted kingfisher	2	22	430	2150	Bobwhite
American kestrel	5	60	430	2150	Bobwhite
Red-cockaded woodpecker[a]	5	51	430	2150	Bobwhite
Mammals					
So. s-tail shrew	9	99	400	2000	Mouse
Red bat	12	120	400	2000	Mouse
E. gray squirrel	2	19	1000	5000	Rat
Meadow vole	8	120	400	2000	Mouse
E. cottontail	1	27	400	2000	Rabbit
White-tailed deer	0.2	4	400	2000	Rabbit
Cotton rat	3	71	1000	5000	Rat
Eastern red fox	0.9	12	1000	5000	Rat
Black bear	0.2	3	1000	5000	Rat
River otter	0.5	5	1000	5000	Rat
Bobcat	1	15	1000	5000	Rat
Amphibians					
Woodhouse toad	4	43	430	2150	Bobwhite
Reptiles					
E. box turtle	2	17	430	2150	Bobwhite
Hognose snake	5	54	430	2150	Bobwhite
Gopher tortoise[b]	0.09	5	430	2150	Bobwhite
Domestic animals					
Cow	0.07	4	400	2000	Rabbit
Chicken	0.8	8	430	2150	Bobwhite
Dog	0.3	3	1000	5000	Rat

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-9

Kerosene wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	10	101	3280[a]	16400	Mallard
Bobwhite quail	9	90	3280	16400	Mallard
Eastern bluebird	21	211	3280	16400	Mallard
Belted kingfisher	7	73	3280	16400	Mallard
American kestrel	17	195	3280	16400	Mallard
Red-cockaded woodpecker[b]	16	164	3280	16400	Mallard
Mammals					
So. s-tail shrew	30	315	5600	28000	Rat
Red bat	36	378	5600	28000	Rat
E. gray squirrel	7	67	5600	28000	Rat
Meadow vole	26	378	5600	28000	Rat
E. cottontail	5	90	5600	28000	Rat
White-tailed deer	0.7	14	5600	28000	Rat
Cotton rat	11	226	5600	28000	Rat
Eastern red fox	3	40	5600	28000	Rat
Black bear	0.8	10	5600	28000	Rat
River otter	2	18	5600	28000	Rat
Bobcat	4	52	5600	28000	Rat
Amphibians					
Woodhouse toad	31	316	3280	16400	Mallard
Reptiles					
E. box turtle	12	125	3280	16400	Mallard
Hognose snake	40	408	3280	16400	Mallard
Gopher tortoise[c]	0.6	20	3280	16400	Mallard
Domestic animals					
Cow	0.3	14	5600	28000	Rat
Chicken	3	30	3280	16400	Mallard
Dog	1	14	5600	28000	Rat

[a] Based on diesel oil LD_{50}.

[b] Federally listed endangered species.

[c] Federally listed threatened species.

Table 8-10

Limonene wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	------------	------------	(mg/kg)	------------	------------
Birds					
Common flicker	3	74	--	--	NA[a]
Bobwhite quail	3	65	--	--	NA
Eastern bluebird	8	159	--	--	NA
Belted kingfisher	3	52	--	--	NA
American kestrel	6	145	--	--	NA
Red-cockaded woodpecker[b]	6	122	--	--	NA
Mammals					
So. s-tail shrew	11	237	1000	5000	Rat
Red bat	14	268	1000	5000	Rat
E. gray squirrel	2	46	1000	5000	Rat
Meadow vole	9	287	1000	5000	Rat
E. cottontail	2	66	1000	5000	Rat
White-tailed deer	0.2	10	1000	5000	Rat
Cotton rat	4	169	1000	5000	Rat
Eastern red fox	1	28	1000	5000	Rat
Black bear	0.2	6	1000	5000	Rat
River otter	0.6	11	1000	5000	Rat
Bobcat	1	37	1000	5000	Rat
Amphibians					
Woodhouse toad	5	102	--	--	NA
Reptiles					
E. box turtle	2	41	--	--	NA
Hognose snake	6	131	--	--	NA
Gopher tortoise[c]	0.1	13	--	--	NA
Domestic animals					
Cow	0.08	10	1000	5000	Rat
Chicken	0.9	20	--	--	NA
Dog	0.4	8	1000	5000	Rat

[a] NA = not available or not applicable.
[b] Federally listed endangered species.
[c] Federally listed threatened species.

Table 8-11

Picloram wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	3	27	400	2000	Pheasant
Bobwhite quail	2	24	400	2000	Pheasant
Eastern bluebird	6	60	400	2000	Pheasant
Belted kingfisher	2	19	400	2000	Pheasant
American kestrel	4	54	400	2000	Pheasant
Red-cockaded woodpecker[a]	4	46	400	2000	Pheasant
Mammals					
So. s-tail shrew	8	89	400	2000	Mouse
Red bat	10	109	400	2000	Mouse
E. gray squirrel	2	16	1640	8200	Rat
Meadow vole	7	109	400	2000	Mouse
E. cottontail	1	24	800	4000	Rabbit
White-tailed deer	0.1	3	144	720	Sheep
Cotton rat	3	63	1640	8200	Rat
Eastern red fox	0.8	10	1640	8200	Rat
Black bear	0.1	2	1640	8200	Rat
River otter	0.4	4	1640	8200	Rat
Bobcat	1	13	1640	8200	Rat
Amphibians					
Woodhouse toad	0.08	2	400	2000	Pheasant
Reptiles					
E. box turtle	0.03	0.9	400	2000	Pheasant
Hognose snake	0.09	2	400	2000	Pheasant
Gopher tortoise[b]	0.008	4	400	2000	Pheasant
Domestic animals					
Cow	0.04	4	144	720	Sheep
Chicken	0.6	7	1200	6000	Chicken
Dog	0.3	3	1640	8200	Rat

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-12

Sulfometuron Methyl wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	0.7	8	1000	5000	Mallard
Bobwhite quail	0.6	7	1000	5000	Mallard
Eastern bluebird	1	16	1000	5000	Mallard
Belted kingfisher	0.5	5	1000	5000	Mallard
American kestrel	1	15	1000	5000	Mallard
Red-cockaded woodpecker[a]	1	13	1000	5000	Mallard
Mammals					
So. s-tail shrew	2	24	1000	5000	Rat
Red bat	3	30	1000	5000	Rat
E. gray squirrel	0.4	5	1000	5000	Rat
Meadow vole	2	30	1000	5000	Rat
E. cottontail	0.3	7	1000	5000	Rat
White-tailed deer	0.04	1	1000	5000	Rat
Cotton rat	0.7	17	1000	5000	Rat
Eastern red fox	0.2	3	1000	5000	Rat
Black bear	0.04	0.7	1000	5000	Rat
River otter	0.1	1	1000	5000	Rat
Bobcat	0.3	4	1000	5000	Rat
Amphibians					
Woodhouse toad	0.9	10	1000	5000	Mallard
Reptiles					
E. box turtle	0.4	4	1000	5000	Mallard
Hognose snake	1	13	1000	5000	Mallard
Gopher tortoise[b]	0.02	1	1000	5000	Mallard
Domestic animals					
Cow	0.02	1	1000	5000	Rat
Chicken	0.2	2	1000	5000	Mallard
Dog	0.08	0.9	1000	5000	Rat

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-13

Tebuthiuron wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	4	123	400	2000	Bobwhite
Bobwhite quail	4	109	400	2000	Bobwhite
Eastern bluebird	9	265	400	2000	Bobwhite
Belted kingfisher	3	87	400	2000	Bobwhite
American kestrel	7	241	400	2000	Bobwhite
Red-cockaded woodpecker[a]	7	204	400	2000	Bobwhite
Mammals					
So. s-tail shrew	12	395	116	579	Mouse
Red bat	15	480	116	579	Mouse
E. gray squirrel	2	76	129	644	Rat
Meadow vole	10	479	116	579	Mouse
E. cottontail	2	109	57	286	Rabbit
White-tailed deer	0.2	16	57	286	Rabbit
Cotton rat	4	282	129	644	Rat
Eastern red fox	1	46	100	500	Dog
Black bear	0.2	11	100	500	Dog
River otter	0.6	19	100	500	Dog
Bobcat	2	62	40	200	Cat
Amphibians					
Woodhouse toad	5	171	340	2000	Bobwhite
Reptiles					
E. box turtle	2	68	400	2000	Bobwhite
Hognose snake	7	218	400	2000	Bobwhite
Gopher tortoise[b]	0.1	22	400	2000	Bobwhite
Domestic animals					
Cow	0.09	17	57	286	Rabbit
Chicken	1	33	100	500	Chicken
Dog	0.5	14	100	500	Dog

[a] Federally listed endangered species.
[b] Federally listed threatened species.

Table 8-14

Triclopyr wildlife and domestic animal doses compared with laboratory acute toxicity

Species	Realistic Dose Estimate	Extreme Dose Estimate	1/5 LD_{50}	LD_{50}	Laboratory Species
	(mg/kg)				
Birds					
Common flicker	16	164	340	1698	Mallard
Bobwhite quail	14	145	340	1698	Mallard
Eastern bluebird	35	353	340	1698	Mallard
Belted kingfisher	12	115	340	1698	Mallard
American kestrel	27	322	340	1698	Mallard
Red-cockaded woodpecker[a]	26	272	340	1698	Mallard
Mammals					
So. s-tail shrew	50	527	94	471	Mouse
Red bat	62	640	94	471	Mouse
E. gray squirrel	10	101	126	630	Rat
Meadow vole	42	639	94	471	Mouse
E. cottontail	7	146	62	310	Guinea pig
White-tailed deer	0.9	22	62	310	Guinea pig
Cotton rat	17	376	126	630	Rat
Eastern red fox	5	62	126	630	Rat
Black bear	1	14	126	630	Rat
River otter	2	25	126	630	Rat
Bobcat	6	82	126	630	Rat
Amphibians					
Woodhouse toad	22	228	340	1698	Mallard
Reptiles					
E. box turtle	9	90	340	1698	Mallard
Hognose snake	29	290	340	1698	Mallard
Gopher tortoise[b]	0.5	29	340	1698	Mallard
Domestic animals					
Cow	0.4	23	62	310	Guinea pig
Chicken	4	44	340	1698	Mallard
Dog	2	19	126	630	Rat

[a] Federally listed endangered species.
[b] Federally listed threatened species.

AQUATIC RISK ANALYSIS

The risks of adverse effects from exposure to herbicides that drift offsite from mechanical ground applications were estimated for the representative aquatic species described in the previous section (see table 7-5). Acute toxicity reference values (LC_{50}'s or EC_{50}'s)[1] and chronic toxicity reference values (MATC's or NOEL's)[1] used in the analysis were selected for the representative species from the summary tables presented in the aquatic hazard analysis (section 6).

In cases where no acute toxicity reference value was available for a representative species, a value was selected from the summary table using the value of the most closely related species. For fish species, preference was given to toxicity values of other species within the same genus or family. If no toxicity values were available for any member of that family, then the lowest value reported for any fish species was used. In the case of 2,4-DP, where values were not available for some species, reference values for 2,4-D butoxyethanol ester were used.

To estimate the risk of adverse effects occurring, the selected toxicity reference values were compared to the typical and maximum estimated environmental concentrations of each herbicide for a body of water 0.61 m (2 ft) deep (see table 7-6). The ratio of the EEC to the LC_{50} (or EC_{50}) is named the quotient value (Q-value). Typical EEC's were based on typical application rates and a distance of 20.1 m (66 ft) from the application site to the body of water. Maximum EEC's were calculated using maximum application rates and a distance of 10.1 m (33 ft) to a water body. EEC's for kerosene were based on the fraction of kerosene in triclopyr ester formulations. The Q-values were compared to the risk criteria proposed by EPA (1986) where the risks of adverse effects to fish or invertebrates are estimated as follows:

Q- value		Risk
EEC/LC_{50}	< 0.1	No acute risk
EEC/LC_{50}	≥ 0.1 and < 0.5	Presumption of risk that may be mitigated
EEC/LC_{50}	≥ 0.5	Presumption of significant risk of acute effects
EEC $<$ NOEL or MATC		No chronic risk

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[1] See Section 6 for definitions of terms.

Table 8-15

Availability of acute toxicity data for invertebrates and aquatic amphibia

Herbicide	Species				
	Crayfish	Water flea	Stonefly-nymph	Virginia oyster	Mudpuppy
2,4-D amine	Yes[a]	Yes	No[b]	No	Yes
2,4-D ester	Yes	Yes	Yes	Yes	No
2,4-DP	Yes	Yes	Yes	Yes	No
Dicamba	No[b]	Yes	No	No	Yes
Diesel fuel	Yes	No	No	No	No
Fosamine	Yes	Yes	No	No	No
Glyphosate-Rodeo	No	Yes	No	No	No
Glyphosate-Roundup	Yes	Yes	Yes	No	No
Hexazinone	Yes	Yes	No	Yes	No
Imazapyr	No	Yes	No	No	No
Kerosene	Yes	No	No	No	No
Limonene	No	No	No	No	No
Picloram and 2,4-D	No	Yes	Yes	Yes	Yes
Sulfometuron methyl	Yes	Yes	No	No	No
Tebuthiuron	Yes	Yes	No	Yes	No
Triclopyr amine	Yes	Yes	No	Yes	No
Triclopyr ester	No	No	No	No	No

[a] Data are available, see tables 6-8 to 6-19 for toxicity reference values.
[b] No data available.

Results of the Risk Analyses

Acute Toxicity

The results of the risk analysis indicate that there is no significant risk of acute adverse effects to any of the representative aquatic species for typical and maximum exposures resulting from drift. All Q-values are less than 0.1. The acute risks to the invertebrates and mudpuppy could not be estimated for some of the chemicals because sufficient toxicity information was not available (see table 8-15). Data were available for Daphnia for all but four of the chemicals; for amphibia, data were available only for 2,4-D amine, dicamba, and picloram (Tordon 101). No data were available for limonene for any aquatic invertebrate or amphibian.

Chronic Toxicity

Very limited information is available on chronic toxicity in aquatic species for most of the chemicals. There are no chronic toxicity data for dicamba, fosamine, Rodeo, Roundup, imazapyr, or limonene; and there are data for only one species for 2,4-D ester, sulfometuron methyl, triclopyr amine, and triclopyr ester. Reasonably good information is available only for 2,4-D amine.

The risks of chronic effects, such as reproductive success or long-term survival, were estimated for those chemicals and species where sufficient information was available. In all of these cases, there was no risk of significant effects (EEC < NOEL or MATC).

In the absence of chronic toxicity information, the likelihood of long-term exposure to herbicide residues was evaluated. The fraction of initial herbicide residue remaining in water was calculated for 1, 2, and 3 weeks after herbicide application using herbicide degradation rates reported in the literature (see table 8-16). Degradation data are not available for limonene. Less than 10 percent of the initial residue remains at 3 weeks for 2,4-D amine, 2,4-D-ester, 2,4-DP, imazapyr, and triclopyr. Residues of approximately 30 percent or greater remain at 3 weeks for fosamine, Rodeo, Roundup, picloram, sulfometuron methyl, and tebuthiuron. Hexazinone has the slowest degradation rate; approximately 63 percent of the initial residue remains after 3 weeks. In streams and other lotic (flowing) waters, herbicide concentrations would quickly be reduced by dilution and transport; however, chronic exposure could occur in ponds and lakes from those herbicides that degrade slowly. For typical conditions, the EEC's for fosamine, Rodeo, hexazinone, picloram, sulfometuron methyl, and tebuthiuron are all at least 10,000 times less than the lowest acute toxicity value (LC_{50} or EC_{50}) reported for each herbicide. It is unlikely that chronic effects would result from these estimated concentrations when there is such a large margin of safety for acute effects. The EEC for the maximum exposure to Roundup is approximately 360 times less than the lowest acute toxicity value. In this case, the risk of chronic effects is probably low because the margin of safety for acute effects is high.

Accidents

EEC's were calculated for a spill of a can containing 19 l (5 gal) of herbicide into a pond and a spill of a helicopter load of 379 l (100 gal) of herbicide mixture into a reservoir (see table 7-6 in section 7). In all cases, the spill into the pond results in higher EEC's than the spill into the reservoir (tables 8-17 through 8-33). No significant acute effects are expected for spills of 2,4-D amine, dicamba, fosamine, Rodeo, hexazinone, imazapyr, picloram + 2,4-D, sulfometuron methyl, tebuthiuron, or triclopyr amine; also, no significant effects are expected from a spill into a reservoir of 2,4-D ester, 2,4-DP, diesel fuel, Roundup, limonene or triclopyr ester (see tables 8-17, 8-20, 8-22, 8-23, 8-25, 8-26, 8-29, 8-30, 8-31, and 8-32). Kerosene is the only chemical expected to have significant acute adverse effects from the reservoir accident. Significant adverse acute effects, including death, would be expected for all representative fish species from a spill into a pond for 2,4-D ester, 2,4-DP, diesel fuel, Roundup, kerosene, limonene, or triclopyr ester (tables 8-18, 8-19, 8-21, 8-24, 8-27, 8-28, and 8-33). Stonefly nymphs

Table 8-16

Fraction of initial herbicide residues remaining in water at weekly intervals

Herbicide	Half-Life[a] (days)	1 Week	2 Weeks	3 Weeks
2,4-D amine	3.0	0.198	0.039	0.008
2,4-D ester	3.0	0.198	0.039	0.008
2,4-DP	6.0	0.446	0.198	0.088
Dicamba	3.0	0.198	0.039	0.008
Diesel fuel	6.0	0.446	0.198	0.088
Fosamine	18.0	0.764	0.583	0.446
Glyphosate-Rodeo	12.0	0.667	0.446	0.297
Glyphosate-Roundup	12.0	0.667	0.446	0.297
Hexazinone	31.3	0.856	0.733	0.628
Imazapyr	4.0	0.297	0.088	0.026
Kerosene	6.0	0.446	0.198	0.088
Limonene	No data	--	--	--
Picloram + 2,4-D	15.0	0.724	0.524	0.379
Sulfomet methyl	14.0	0.707	0.500	0.354
Tebuthiuron	17.5	0.758	0.574	0.435
Triclopyr amine	0.4	0.00001	$9x10^{-11}$	$9.0x10^{-16}$
Triclopyr ester	0.4	0.00001	$9x10^{-11}$	$9.0x10^{-16}$

[a] Sources: American Cyanamid Co. (1986); Dennis et al. (1977); Ghassemi et al. (1981); Han (1979); Harvey et al. (1985); Mabey et al. (1982); Rhodes (1980); USDA (1984); USDA (1986).

also would be adversely affected from spills of 2,4-D ester and 2,4-DP (tables 8-18 and 8-19). No significant effects are expected for those invertebrates where there is sufficient toxicity information to estimate risk (see table 8-15 for data gaps).

Estimated herbicide concentrations in a body of water that is accidentally directly sprayed at typical application rates are greater than those estimated for the reservoir-spill. The EEC's are less than those estimated for the pond spill, except for 2,4-D + picloram, where the EEC's are approximately equal. At maximum application rates, the EEC's for direct spraying are greater than the EEC's for the reservoir spill, and they are greater than the EEC's for the pond spill for dicamba, fosamine, hexazinone, picloram + 2,4-D, and triclopyr amine.

In general, the risk to aquatic species is the same for the scenarios of direct spraying at maximum rates and the pond spill, with the exceptions of limonene and sulfometuron methyl in which risk from direct spraying is less. Aquatic species exposed to limonene as a result of direct spraying are at "slight" risk rather than the "significant" risk from a spill to a pond (see table 8-28). Exposure to sulfometuron methyl as a result of direct spraying results in Q-values indicating no significant risk compared to "slight" risk from a pond spill (table 8-30).

POTENTIAL EFFECTS ON THREATENED OR ENDANGERED SPECIES

Federal policies and procedures for protecting threatened and endangered species of fish, wildlife, and plants were established by the Endangered Species Act of 1973 (16 U.S.C. 1531 et seq.) and regulations issued pursuant to the act. The purposes of the act are to provide mechanisms for conservation of threatened and endangered species and the habitats upon which they depend, and to achieve the goals of international treaties and conventions related to endangered species. Under the act, the Secretary of the Interior is required to determine which species are threatened or endangered and to issue regulations for the protection of those species.

There are a number of threatened and endangered species on National Forest lands in Region 8. Three of those species were selected for analysis of potential impacts of Region 8 herbicide programs: the red-cockaded woodpecker (Picoides borealis), the smoky madtom (Noturus baileyi), and the gopher tortoise (Gopherus polyphemus).

Analysis of potential effects on these species must include consideration of the potential for the species to be exposed to herbicides either directly or through their food supply. Potential herbicide exposures of the red-cockaded woodpecker and gopher tortoise were estimated in the analysis of terrestrial wildlife. Exposures of the smoky madtom were estimated in the aquatic species risk analysis.

Red-Cockaded Woodpecker

Red-cockaded woodpeckers forage in mature pines by gleaning insects from the bark of trees or using the beak and tongue to remove insects from bark crevices. Mast and fruits may form a minor food source (Scott et al., 1977).

Herbicides may affect the red-cockaded woodpecker directly through oral or dermal doses as was shown for other wildlife species in this risk assessment. Two herbicides that appear to present a significant potential for direct toxic effects when applied to the woodpeckers' foraging or nesting areas at greater than typical application rates are 2,4-D and 2,4-DP. While aerial and ground mechanical applications of these two herbicides can pose a serious threat to the birds, hand applications should not. The remaining herbicides present a moderately low to very low potential for toxic effects, even when it is assumed that the red-cockaded woodpeckers receive a direct spraying and feed exclusively on contaminated insects.

Gopher Tortoise

The gopher tortoise is found primarily in well-drained habitats, particularly in the sandhills and longleaf pine-turkey oak associations of the Southeast where it feeds on herbaceous vegetation under open tree canopies. It occurs in the Ocala, Osceola, Apalachicola, Conecuh, and DeSoto National Forests (Seehorn, 1982).

Maintaining herbaceous growth, by keeping an open tree canopy, and protecting burrows are essential in the recovery of the gopher tortoise.

Because of its low metabolic rate and heavy carapace, the only significant route of herbicide intake by the gopher tortoise is its food. Earlier in the wildlife risk assessment it was shown that even in the extreme case the tortoise is not likely to receive a toxic dose of any of the 14 herbicides and additives evaluated.

Smoky Madtom

The smoky madtom is federally classified as an endangered fish species. It is found only within 10.5 km (6.5 mi) of Citico Creek, a tributary of the Little Tennessee River in Monroe County, Tennessee (Cindy Witkowski, U.S. Forest Service, personal communication, 1987). The madtom is probably a nocturnal insectivore, although little information is available on the fish's life history. The limited distribution of this species makes it highly vulnerable to extinction through any alteration of its habitat.

The potential for contamination of the smoky madtom's critical habitat resulting from spraying of herbicides for vegetation management was evaluated. EPA uses a criterion of less than 1/20 of the lowest reported aquatic LC_{50} as a safe (minimal risk) acute exposure level for an endangered fish species. This criterion was used to estimate risk to the smoky madtom. The results indicate that there is no risk (Q values are all less than 0.05) to the smoky madtom using typical application rates (see table 8-34). The same table also shows that no significant risk exists even when exposures are evaluated for maximum herbicide application rates and minimum buffer zones (table 8-34).

Table 8-17

Risk analysis for 2,4-D amine for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.7 ppm		
Rainbow trout	>100	<0.02	No risk
Brook trout	>100	<0.02	No risk
Largemouth bass	236	0.007	No risk
Smallmouth bass	236	0.007	No risk
Bluegill	168	0.01	No risk
Green sunfish	168	0.01	No risk
Fathead minnow	335	0.005	No risk
Gizzard shad	>100	<0.02	No risk
Northern hogsucker	>100	<0.02	No risk
Mosquitofish	405	0.004	No risk
Chain pickerel	>100	<0.02	No risk
Crayfish	>100	<0.02	No risk
Water flea	4	0.4	Slight[b]
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	200	0.009	No risk
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.12 ppm		
Rainbow trout	>100	<0.001	No risk
Brook trout	>100	<0.001	No risk
Largemouth bass	236	0.0005	No risk
Smallmouth bass	236	0.0005	No risk
Bluegill	168	0.0007	No risk
Green sunfish	168	0.0007	No risk
Fathead minnow	335	0.0003	No risk
Gizzard shad	>100	<0.001	No risk
Northern hogsucker	>100	<0.001	No risk
Mosquitofish	405	0.0003	No risk
Chain pickerel	>100	<0.001	No risk
Crayfish	>100	<0.001	No risk
Water flea	4	0.03	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	200	0.0006	No risk

[a] Based on EPA (1986).

[b] Presumption of risk that may be mitigated according to EPA risk criteria.

Table 8-18

Risk analysis for 2,4-D ester for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.7 ppm		
Rainbow trout	1.49	1.0	Significant
Brook trout	1.49	1.0	Significant
Largemouth bass	1.2	1.0	Significant
Smallmouth bass	1.2	1.0	Significant
Bluegill	1.2	1.0	Significant
Green sunfish	1.2	1.0	Significant
Fathead minnow	3.3	0.5	Significant
Gizzard shad	1.2	1.0	Significant
Northern hogsucker	1.2	1.0	Significant
Mosquitofish	1.2	1.0	Significant
Chain pickerel	1.2	1.0	Significant
Crayfish	>100.0	0.02	No risk
Water flea	5.6	0.3	Slight[b]
Stonefly nymph	1.6	1.0	Significant
Virginia oyster	3.75	0.5	Slight[b]
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.17 ppm		
Rainbow trout	1.49	0.1	Slight
Brook trout	1.49	0.1	Slight
Largemouth bass	1.2	0.1	Slight
Smallmouth bass	1.2	0.1	Slight
Bluegill	1.2	0.1	Slight
Green sunfish	1.2	0.1	Slight
Fathead minnow	3.3	0.05	No risk
Gizzard shad	1.2	0.1	Slight
Northern hogsucker	1.2	0.1	Slight
Mosquitofish	1.2	0.1	Slight
Chain pickerel	1.2	0.1	Slight
Crayfish	>100.0	<0.002	No risk
Water flea	5.6	0.03	No risk
Stonefly nymph	1.6	0.1	Slight
Virginia oyster	3.75	0.05	No risk
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

[a] Presumption of risk that may be mitigated according to EPA risk criteria.

Table 8-19

Risk analysis for 2,4-DP for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.7 ppm		
Rainbow trout	1.42[b]	1.0	Significant
Brook trout	1.42[b]	1.0	Significant
Largemouth bass	1.1	2.0	Significant
Smallmouth bass	1.1	2.0	Significant
Bluegill	1.1	2.0	Significant
Green sunfish	1.1	2.0	Significant
Fathead minnow	3.3[b]	0.5	Significant
Gizzard shad	1.2[b]	1.0	Significant
Northern hogsucker	1.5	1.0	Significant
Mosquitofish	1.2[b]	1.0	Significant
Chain pickerel	1.2[b]	1.0	Significant
Crayfish	>100.0[b]	<0.02	No risk
Water flea	5.6[b]	0.3	Slight[c]
Stonefly nymph	1.6[b]	1.0	Significant
Virginia oyster	3.75[b]	0.5	Slight[c]
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.14 ppm		
Rainbow trout	1.42	0.1	Slight
Brook trout	1.42	0.1	Slight
Largemouth bass	1.1	0.1	Slight
Smallmouth bass	1.1	0.1	Slight
Bluegill	1.1	0.1	Slight
Green sunfish	1.1	0.1	Slight
Fathead minnow	3.3	0.04	No risk
Gizzard shad	1.2	0.1	Slight
Northern hogsucker	1.5	0.1	No risk
Mosquitofish	1.2	0.1	Slight
Chain pickerel	1.2	0.1	Slight
Crayfish	>100.0	<0.001	No risk
Water flea	5.6	0.03	No risk
Stonefly nymph	1.6	0.09	No risk
Virginia oyster	3.75	0.04	No risk
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

[b] Based on 2,4-D ester toxicity value because of limited information available for 2,4-DP.

[c] Presumption of risk that may be mitigated according to EPA risk criteria.

Table 8-20

Risk analysis for dicamba for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 0.46 ppm		
Rainbow trout	28	0.02	No risk
Brook trout	28	0.02	No risk
Largemouth bass	28	0.02	No risk
Smallmouth bass	28	0.02	No risk
Bluegill	>50	<0.009	No risk
Green sunfish	28	0.02	No risk
Fathead minnow	28	0.02	No risk
Gizzard shad	28	0.02	No risk
Northern hogsucker	28	0.02	No risk
Mosquitofish	28	0.02	No risk
Chain pickerel	28	0.02	No risk
Crayfish	--	--	No data
Water flea	11	0.04	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	106	0.004	No risk
	No aerial use		

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[a] Based on EPA (1986).

Table 8-21

Risk analysis for diesel fuel for accidents

Representative Species	LC$_{50}$ or EC$_{50}$ (ppm)	Q-Value (EEC/LC$_{50}$)	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 3.1 ppm		
Rainbow trout	>0.19	<20.0	Significant
Brook trout	>0.19	<20.0	Significant
Largemouth bass	>0.19	<20.0	Significant
Smallmouth bass	>0.19	<20.0	Significant
Bluegill	>0.19	<20.0	Significant
Green sunfish	>0.19	<20.0	Significant
Fathead minnow	>0.19	<20.0	Significant
Gizzard shad	>0.19	<20.0	Significant
Northern hogsucker	>0.19	<20.0	Significant
Mosquitofish	>0.19	<20.0	Significant
Chain pickerel	>0.19	<20.0	Significant
Crayfish	14.1	0.2	Slight[b]
Water flea	--	--	No data
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.043 ppm		
Rainbow trout	>0.19	<0.2	Slight[b]
Brook trout	>0.19	<0.2	Slight[b]
Largemouth bass	>0.19	<0.2	Slight[b]
Smallmouth bass	>0.19	<0.2	Slight[b]
Bluegill	>0.19	<0.2	Slight[b]
Green sunfish	>0.19	<0.2	Slight[b]
Fathead minnow	>0.19	<0.2	Slight[b]
Gizzard shad	>0.19	<0.2	Slight[b]
Northern hogsucker	>0.19	<0.2	Slight[b]
Mosquitofish	>0.19	<0.2	Slight[b]
Chain pickerel	>0.19	<0.2	Slight[b]
Crayfish	14.1	0.003	No Risk
Water flea	--	--	No data
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

[b] Presumption of risk that may be mitigated according to EPA risk criteria.

Table 8-22

Risk analysis for fosamine for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.8 ppm		
Rainbow trout	>100	<0.02	No risk
Brook trout	>100	<0.02	No risk
Largemouth bass	670	0.003	No risk
Smallmouth bass	670	0.003	No risk
Bluegill	670	0.003	No risk
Green sunfish	670	0.003	No risk
Fathead minnow	>1,000	<0.002	No risk
Gizzard shad	>100	<0.02	No risk
Northern hogsucker	>100	<0.02	No risk
Mosquitofish	>100	<0.02	No risk
Chain pickerel	>100	<0.02	No risk
Crayfish	3,547	0.0005	No risk
Water flea	1,524	0.001	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.29 ppm		
Rainbow trout	>100	<0.002	No risk
Brook trout	>100	<0.003	No risk
Largemouth bass	670	0.0004	No risk
Smallmouth bass	670	0.0004	No risk
Bluegill	670	0.0004	No risk
Green sunfish	670	0.0004	No risk
Fathead minnow	>1,000	<0.0003	No risk
Gizzard shad	>100	<0.003	No risk
Northern hogsucker	>100	<0.003	No risk
Mosquitofish	>100	<0.003	No risk
Chain pickerel	>100	<0.003	No risk
Crayfish	3,547	0.00008	No risk
Water flea	1,524	0.0002	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

Table 8-23

Risk analysis for glyphosate, Rodeo formulation, for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.4 ppm		
Rainbow trout	>1,000	<0.001	No risk
Brook trout	>1,000	<0.001	No risk
Largemouth bass	>1,000	<0.001	No risk
Smallmouth bass	>1,000	<0.001	No risk
Bluegill	>1,000	<0.001	No risk
Green sunfish	>1,000	<0.001	No risk
Fathead minnow	>1,000	<0.001	No risk
Gizzard shad	>1,000	<0.001	No risk
Northern hogsucker	>1,000	<0.001	No risk
Mosquitofish	>1,000	<0.001	No risk
Chain pickerel	>1,000	<0.001	No risk
Crayfish	--	--	No data
Water flea	930	0.001	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = .09 ppm		
Rainbow trout	>1,000	<0.0001	No risk
Brook trout	>1,000	<0.0001	No risk
Largemouth bass	>1,000	<0.0001	No risk
Smallmouth bass	>1,000	<0.0001	No risk
Bluegill	>1,000	<0.0001	No risk
Green sunfish	>1,000	<0.0001	No risk
Fathead minnow	>1,000	<0.0001	No risk
Gizzard shad	>1,000	<0.0001	No risk
Northern hogsucker	>1,000	<0.0001	No risk
Mosquitofish	>1,000	<0.0001	No risk
Chain pickerel	>1,000	<0.0001	No risk
Crayfish	--	--	No data
Water flea	930	0.0001	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

Table 8-24

Risk analysis for glyphosate, Roundup formulation, for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.4 ppm		
Rainbow trout	1.3	1.0	Significant
Brook trout	1.3	1.0	Significant
Largemouth bass	1.8	0.8	Significant
Smallmouth bass	1.8	0.8	Significant
Bluegill	1.8	0.8	Significant
Green sunfish	1.8	0.8	Significant
Fathead minnow	2.3	0.6	Significant
Gizzard shad	1.3	1.0	Significant
Northern hogsucker	1.3	1.0	Significant
Mosquitofish	1.3	1.0	Significant
Chain pickerel	1.3	1.0	Significant
Crayfish	>1,000	<0.001	No risk
Water flea	3	0.5	Slight[b]
Stonefly nymph	10	0.1	Slight[b]
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = .09 ppm		
Rainbow trout	1.3	0.1	Slight[b]
Brook trout	1.3	0.1	Slight[b]
Largemouth bass	1.8	0.1	No risk
Smallmouth bass	1.8	0.1	No risk
Bluegill	1.8	0.1	No risk
Green sunfish	1.8	0.1	No risk
Fathead minnow	2.3	0.08	No risk
Gizzard shad	1.3	0.1	Slight[b]
Northern hogsucker	1.3	0.1	Slight[b]
Mosquitofish	1.3	0.1	Slight[b]
Chain pickerel	1.3	0.1	Slight[b]
Crayfish	>1,000	<0.0002	No risk
Water flea	3	0.06	No risk
Stonefly nymph	10	0.02	No risk
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

[b] Presumption of risk that may be mitigated according to EPA risk criteria.

Table 8-25

Risk analysis for hexazinone for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 0.92 ppm		
Rainbow trout	>180	<0.005	No risk
Brook trout	>100	<0.009	No risk
Largemouth bass	370	0.002	No risk
Smallmouth bass	370	0.002	No risk
Bluegill	370	0.002	No risk
Green sunfish	370	0.002	No risk
Fathead minnow	274	0.003	No risk
Gizzard shad	>100	<0.009	No risk
Northern hogsucker	>100	<0.009	No risk
Mosquitofish	>100	<0.009	No risk
Chain pickerel	>100	<0.009	No risk
Crayfish	>1,000	<0.0009	No risk
Water flea	151.6	0.006	No risk
Stonefly nymph	--	--	No data
Virginia oyster	320	0.003	No risk
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.086 ppm		
Rainbow trout	>180	<0.0005	No risk
Brook trout	>100	<0.0009	No risk
Largemouth bass	370	0.0002	No risk
Smallmouth bass	370	0.0002	No risk
Bluegill	370	0.0002	No risk
Green sunfish	370	0.0002	No risk
Fathead minnow	274	0.0003	No risk
Gizzard shad	>100	0.0009	No risk
Northern hogsucker	>100	<0.0009	No risk
Mosquitofish	>100	<0.0009	No risk
Chain pickerel	>100	<0.0009	No risk
Crayfish	>1,000	<0.00009	No risk
Water flea	151.6	0.0006	No risk
Stonefly nymph	--	--	No data
Virginia oyster	320	0.0003	No risk
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

Table 8-26

Risk analysis for imazapyr for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 0.92 ppm		
Rainbow trout	110	0.008	No risk
Brook trout	110	0.008	No risk
Largemouth bass	>180	<0.005	No risk
Smallmouth bass	>180	<0.005	No risk
Bluegill	>180	<0.005	No risk
Green sunfish	>180	<0.005	No risk
Fathead minnow	110	0.008	No risk
Gizzard shad	110	0.008	No risk
Northern hogsucker	110	0.008	No risk
Mosquitofish	110	0.008	No risk
Chain pickerel	110	0.008	No risk
Crayfish	--	--	No data
Water flea	>350	<0.003	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.043 ppm		
Rainbow trout	110	0.0004	No risk
Brook trout	110	0.0004	No risk
Largemouth bass	>180	<0.0002	No risk
Smallmouth bass	>180	<0.0002	No risk
Bluegill	>180	<0.0002	No risk
Green sunfish	>180	<0.0002	No risk
Fathead minnow	110	0.0004	No risk
Gizzard shad	110	0.0004	No risk
Northern hogsucker	110	0.0004	No risk
Mosquitofish	110	0.0004	No risk
Chain pickerel	110	0.0004	No risk
Crayfish	--	--	No data
Water flea	>350	<0.0001	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

Table 8-27

Risk analysis for kerosene for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.0 ppm		
Rainbow trout	>0.19	<5.0	Significant
Brook trout	>0.19	<5.0	Significant
Largemouth bass	>0.19	<5.0	Significant
Smallmouth bass	>0.19	<5.0	Significant
Bluegill	>0.19	<5.0	Significant
Green sunfish	>0.19	<5.0	Significant
Fathead minnow	>0.19	<5.0	Significant
Gizzard shad	>0.19	<5.0	Significant
Northern hogsucker	>0.19	<5.0	Significant
Mosquitofish	>0.19	<5.0	Significant
Chain pickerel	>0.19	<5.0	Significant
Crayfish	14.1	0.07	No risk
Water flea	--	--	No data
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.13 ppm		
Rainbow trout	>0.19	<0.7	Significant
Brook trout	>0.19	<0.7	Significant
Largemouth bass	>0.19	<0.7	Significant
Smallmouth bass	>0.19	<0.7	Significant
Bluegill	>0.19	<0.7	Significant
Green sunfish	>0.19	<0.7	Significant
Fathead minnow	>0.19	<0.7	Significant
Gizzard shad	>0.19	<0.7	Significant
Northern hogsucker	>0.19	<0.7	Significant
Mosquitofish	>0.19	<0.7	Significant
Chain pickerel	>0.19	<0.7	Significant
Crayfish	14.1	0.009	No risk
Water flea	--	--	No data
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

Table 8-28

Risk analysis for limonene for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 3.3 ppm		
Rainbow trout	5.2	0.6	Significant
Brook trout	5.2	0.6	Significant
Largemouth bass	5.2	0.6	Significant
Smallmouth bass	5.2	0.6	Significant
Bluegill	5.2	0.6	Significant
Green sunfish	5.2	0.6	Significant
Fathead minnow	5.2	0.6	Significant
Gizzard shad	5.2	0.6	Significant
Northern hogsucker	5.2	0.6	Significant
Mosquitofish	5.2	0.6	Significant
Chain pickerel	5.2	0.6	Significant
Crayfish	--	--	No data
Water flea	--	--	No data
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.052 ppm		
Rainbow trout	5.2	0.01	No risk
Brook trout	5.2	0.01	No risk
Largemouth bass	5.2	0.01	No risk
Smallmouth bass	5.2	0.01	No risk
Bluegill	5.2	0.01	No risk
Green sunfish	5.2	0.01	No risk
Fathead minnow	5.2	0.01	No risk
Gizzard shad	5.2	0.01	No risk
Northern hogsucker	5.2	0.01	No risk
Mosquitofish	5.2	0.01	No risk
Chain pickerel	5.2	0.01	No risk
Crayfish	--	--	No data
Water flea	--	--	No data
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

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[a] Based on EPA (1986).

Table 8-29

Risk analysis for picloram + 2,4-D mixture for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 0.12 ppm		
Rainbow trout	40.4	0.003	No risk
Brook trout	64.9	0.002	No risk
Largemouth bass	40.4	0.003	No risk
Smallmouth bass	40.4	0.003	No risk
Bluegill	40.4	0.003	No risk
Green sunfish	40.4	0.003	No risk
Fathead minnow	17.4	0.007	No risk
Gizzard shad	17.4	0.007	No risk
Northern hogsucker	17.4	0.007	No risk
Mosquitofish	17.4	0.007	No risk
Chain pickerel	17.4	0.007	No risk
Crayfish	--	--	No data
Water flea	380	0.0003	No risk
Stonefly nymph	48	0.003	No risk
Virginia oyster	380	0.0003	No risk
Mudpuppy	95	0.001	No risk
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.43 ppm		
Rainbow trout	40.4	0.001	No risk
Brook trout	64.9	0.0007	No risk
Largemouth bass	40.4	0.001	No risk
Smallmouth bass	40.4	0.001	No risk
Bluegill	40.4	0.001	No risk
Green sunfish	40.4	0.001	No risk
Fathead minnow	17.4	0.002	No risk
Gizzard shad	17.4	0.002	No risk
Northern hogsucker	17.4	0.002	No risk
Mosquitofish	17.4	0.002	No risk
Chain pickerel	17.4	0.002	No risk
Crayfish	--	--	No data
Water flea	380	0.0001	No risk
Stonefly nymph	48	0.0009	No risk
Virginia oyster	380	0.0001	No risk
Mudpuppy	95	0.0005	No risk

[a] Based on EPA (1986).

Table 8-30

Risk analysis for sulfometuron methyl for accidents

Representative Species	LC$_{50}$ or EC$_{50}$ (ppm)	Q-Value (EEC/LC$_{50}$)	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.6 ppm		
Rainbow trout	>12.5	<0.1	Slight[b]
Brook trout	>12.5	<0.1	Slight[b]
Largemouth bass	>12.5	<0.1	Slight[b]
Smallmouth bass	>12.5	<0.1	Slight[b]
Bluegill	>12.5	<0.1	Slight[b]
Green sunfish	>12.5	<0.1	Slight[b]
Fathead minnow	>12.5	<0.1	Slight[b]
Gizzard shad	>12.5	<0.1	Slight[b]
Northern hogsucker	>12.5	<0.1	Slight[b]
Mosquitofish	>12.5	<0.1	Slight[b]
Chain pickerel	>12.5	<0.1	Slight[b]
Crayfish	>5,000	<0.0003	No risk
Water flea	>12.5	<0.1	Slight[b]
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.012 ppm		
Rainbow trout	12.5	0.0009	No risk
Brook trout	12.5	0.0009	No risk
Largemouth bass	12.5	0.0009	No risk
Smallmouth bass	12.5	0.0009	No risk
Bluegill	12.5	0.0009	No risk
Green sunfish	12.5	0.0009	No risk
Fathead minnow	12.5	0.0009	No risk
Gizzard shad	12.5	0.0009	No risk
Northern hogsucker	12.5	0.0009	No risk
Mosquitofish	12.5	0.0009	No risk
Chain pickerel	12.5	0.0009	No risk
Cratfish	>5,000	<0.000002	No risk
Water flea	>12.5	<0.0009	No risk
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

[b] Presumption of risk that may be mitigated according to EPA risk criteria.

Table 8-31

Risk analysis for tebuthiuron for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 3.7 ppm		
Rainbow trout	144	0.03	No risk
Brook trout	144	0.03	No risk
Largemouth bass	112	0.03	No risk
Smallmouth bass	112	0.03	No risk
Bluegill	112	0.03	No risk
Green sunfish	112	0.03	No risk
Fathead minnow	>160	<0.02	No risk
Gizzard shad	112	0.03	No risk
Northern hogsucker	112	0.03	No risk
Mosquitofish	112	0.03	No risk
Chain pickerel	112	0.03	No risk
Crayfish	>320	<0.01	No risk
Water flea	297	0.01	No risk
Stonefly nymph	--	--	No data
Virginia oyster	180	0.02	No risk
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.17 ppm		
Rainbow trout	144	0.001	No risk
Brook trout	144	0.001	No risk
Largemouth bass	112	0.002	No risk
Smallmouth bass	112	0.002	No risk
Bluegill	112	0.002	No risk
Green sunfish	112	0.002	No risk
Fathead minnow	>160	<0.001	No risk
Gizzard shad	112	0.002	No risk
Northern hogsucker	112	0.002	No risk
Mosquitofish	112	0.002	No risk
Chain pickerel	112	0.002	No risk
Crayfish	>320	<0.0005	No risk
Water flea	297	0.0006	No risk
Stonefly nymph	--	--	No data
Virginia oyster	180	0.001	No risk
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

Table 8-32

Risk analysis for triclopyr amine for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 0.12 ppm		
Rainbow trout	552	0.003	No risk
Brook trout	552	0.003	No risk
Largemouth bass	891	0.002	No risk
Smallmouth bass	891	0.002	No risk
Bluegill	891	0.002	No risk
Green sunfish	891	0.002	No risk
Fathead minnow	120	0.01	No risk
Gizzard shad	120	0.01	No risk
Northern hogsucker	120	0.01	No risk
Mosquitofish	120	0.01	No risk
Chain pickerel	120	0.01	No risk
Crayfish	>1,000	<0.001	No risk
Water flea	1,170	0.001	No risk
Stonefly nymph	--	--	No data
Virginia oyster	56	0.02	No risk
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.17 ppm		
Rainbow trout	552	0.0003	No risk
Brook trout	552	0.0003	No risk
Largemouth bass	891	0.0002	No risk
Smallmouth bass	891	0.0002	No risk
Bluegill	891	0.0002	No risk
Green sunfish	891	0.0002	No risk
Fathead minnow	120	0.001	No risk
Gizzard shad	120	0.001	No risk
Northern hogsucker	120	0.001	No risk
Mosquitofish	120	0.001	No risk
Chain pickerel	120	0.001	No risk
Crayfish	>1,000	<0.0002	No risk
Water flea	1,170	0.0001	No risk
Stonefly nymph	--	--	No data
Virginia oyster	56	0.003	No risk
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

Table 8-33

Risk analysis for triclopyr ester for accidents

Representative Species	LC_{50} or EC_{50} (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
	19 liter (5 gallon) drum spill into pond EEC = 1.8 ppm		
Rainbow trout	0.74	2.0	Significant
Brook trout	0.74	2.0	Significant
Largemouth bass	0.87	2.0	Significant
Smallmouth bass	0.87	2.0	Significant
Bluegill	0.87	2.0	Significant
Green sunfish	0.87	2.0	Significant
Fathead minnow	0.74	2.0	Significant
Gizzard shad	0.74	2.0	Significant
Northern hogsucker	0.74	2.0	Significant
Mosquitofish	0.74	2.0	Significant
Chain pickerel	0.74	2.0	Significant
Crayfish	--	--	No data
Water flea	--	--	No data
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data
	379 liter (100 gallon) aerial spill into reservoir, EEC = 0.23 ppm		
Rainbow trout	0.74	0.3	Slight[b]
Brook trout	0.74	0.3	Slight[b]
Largemouth bass	0.87	0.3	Slight[b]
Smallmouth bass	0.87	0.3	Slight[b]
Bluegill	0.87	0.3	Slight[b]
Green sunfish	0.87	0.3	Slight[b]
Fathead minnow	0.74	0.3	Slight[b]
Gizzard shad	0.74	0.3	Slight[b]
Northern hogsucker	0.74	0.3	Slight[b]
Mosquitofish	0.74	0.3	Slight[b]
Chain pickerel	0.74	0.3	Slight[b]
Crayfish	--	--	No data
Water flea	--	--	No data
Stonefly nymph	--	--	No data
Virginia oyster	--	--	No data
Mudpuppy	--	--	No data

[a] Based on EPA (1986).

[b] Presumption of risk that may be mitigated according to EPA risk criteria.

Table 8-34

Acute risk to endangered fish species--Smoky madtom--
under routine conditions

Herbicide	Lowest LC_{50} (ppm)	EEC (ppm)	Q-Value (EEC/LC_{50})	Risk Level[a]
		Typical		
2,4-D amine	4.0	0.0016	0.0004	No risk
2,4-D ester	0.44	0.0025	0.006	No risk
2,4-DP	0.44[b]	0.0025	0.006	No risk
Dicamba	11	0.0013	0.0001	No risk
Diesel fuel	>0.19	0.0013	<0.007	No risk
Fosamine	100	0.0049	0.00005	No risk
Glyphosate-Rodeo	930	0.0010	0.000001	No risk
Glyphosate-Roundup	1.3	0.0010	0.0008	No risk
Hexazinone	56	0.0011	0.00002	No risk
Imazapyr	100	0.00048	0.000005	No risk
Kerosene	>0.19	0.0014	<0.008	No risk
Limonene	5.2	0.00057	0.0001	No risk
Picloram and 2,4-D	17.4	0.00044	0.00003	No risk
Sulfometuron methyl	>12.5	0.00011	<0.000009	No risk
Tebuthiuron	48	0.0002	0.000004	No risk
Triclopyr amine	101	0.0025	0.00003	No risk
Triclopyr ester	0.74	0.0025	0.003	No risk
		Maximum		
2,4-D amine	4.0	0.0036	0.0009	No risk
2,4-D ester	0.44	0.0063	0.01	No risk
2,4-DP	0.44[b]	0.0054	0.01	No risk
Dicamba	11	0.0027	0.0002	No risk
Diesel fuel	>0.19	0.0031	<0.02	No risk
Fosamine	100	0.011	0.0001	No risk
Glyphosate-Rodeo	930	0.0036	0.000004	No risk
Glyphosate-Roundup	1.3	0.0036	0.003	No risk
Hexazinone	56	0.0054	0.0001	No risk
Imazapyr	100	0.0013	0.0001	No risk
Kerosene	>0.19	0.0041	<0.02	No risk
Limonene	5.2	0.0032	0.0006	No risk
Picloram and 2,4-D	17.4	0.0013	0.00007	No risk
Sulfometuron methyl	>12.5	0.00033	<0.00003	No risk
Tebuthiuron	48	0.0023	0.00005	No risk
Triclopyr amine	101	0.0072	0.00007	No risk
Triclopyr ester	0.74	0.0072	0.01	No risk

[a] Based on EPA (1986).

[b] The lowest LC_{50} for 2,4-D butoxyethanol ester is used because of limited toxicity information available for 2,4-DP.

Table 8-35

Species' common and scientific names

Common Name	Scientific Name
Birds	
Common flicker	*Colaptes auratus*
Bobwhite quail	*Colinus virginianus*
Eastern bluebird	*Sialia sialis*
Belted kingfisher	*Megaceryle alcyon*
American kestrel	*Falco sparvarius*
Red-cockaded woodpecker	*Picoides berealis*
Black-capped chickadee	*Parus atricapillus*
Bobwhite quail	*Colinus virginianus*
Cardinal	*Cardinalis cardinalis*
Domestic chicken	*Gallus gallus*
Chukar (partridge)	*Alectoris chukar*
Downy woodpecker	*Picoides pubescens*
Japanese quail	*Coturnix japonica*
Mallard	*Anas platyrhynchos*
Domestic pigeon (rock dove)	*Columbia livia*
Ring-necked pheasant	*Phasianus colchicus*
Rose-breasted grosbeak	*Pheucitus ludovicianus*
Song sparrow	*Melospiza melodia*
White-breasted nuthatch	*Sitta carolinensis*
Mammals	
Southern short-tailed shrew	*Blarina carolinensis*
Red bat	*Lasiurus borealis*
Eastern gray squirrel	*Sciurus carolinensis*
Pine vole	*Microtus pinetorum*
Eastern cottontail	*Sylvilagus floridanus*
White-tailed deer	*Odocoileus virginianus*
Domestic cow	*Bos taurus*
Cotton rat	*Sigmodon hispidus*
Eastern red fox	*Vulpes fulva*
Black bear	*Ursus americanus*
River otter	*Lutra canadensis*
Bobcat	*Lynx rufus*
Domestic cat	*Felis domesticus*
Cottontail rabbit	*Sylvilagus floridanus*
Domestic dog	*Canis familiaris*
Fallow deer	*Dama dama*
Guinea pig	*Cavia cobaya*
Rhesus monkey	*Macaca rhesus*
Moose	*Alces alces*
House mouse	*Mus musculus*

Table 8-35 (continued)

Species' common and scientific names

Common Name	Scientific Name
Mule deer	*Odocoileus hemionus hemionus*
Opossum	*Didelphis virginiana*
Horse (pony)	*Equis caballus*
Prairie vole	*Microtus ochrogaster*
Domestic rabbit	*Oryctolagus cuniculus*
Raccoon	*Procyon lotor*
Albino rat	*Rattus* spp.
Red deer	*Cervus elaphus*
Reindeer	*Rangifer tarandus*
Roedeer	*Capreolus capreolus*
Sheep	*Ovis aries*
Skunk	*Mephitis mephitis*
Swine	*Sus scrofa*
White-tailed deer	*Odocoileus virginianus*
Amphibians	
Woodhouse toad	*Bufo woodhousei*
Mudpuppy	*Necturus maculosus*
Frog[a]	*Adelotus brevis*
Frog[a]	*Lymnodynastes peroni*
Giant toad	*Bufo marinus*
Reptiles	
Eastern box turtle	*Terrapene carolina*
Gopher tortoise	*Gopherus polyphemus*
Hognose snake	*Heterodon platyrhinos*
Indigo snake	*Drymarchon corais*
Invertebrates	
Earthworm	*Lumbricus sp.*
American bird grasshopper	*Schistocerca americana*
Leafcutting ant	*Atta texana*
Honey bees	*Apis melliferu*
Honey bee (referred to in text as bees)	*Apis melifera*
Aquatic invertebrates	
Blue crab	*Callinectes sapidus*
Copepoda[a]	*Nitocra spinipes*
Crayfish	*Orconectes nais*
Crayfish	*Procambarus* sp.
Eastern or Virginia oyster	*Crassostrea virginica*

Table 8-35 (continued)

Species' common and scientific names

Common Name	Scientific Name
Fiddler crab	*Uca pugilator*
Glass shrimp	*Palaemonetes kadiakensis*
Grass shrimp	*Palaemonetes pugio*
Mayfly[a]	*Ephemerella walkeri*
Midge[a]	*Chironomus plumosus*
Pink shrimp	*Penaeus duorarum*
Scud[a]	*Gammarus* sp.
Seed shrimp	*Cypridopsis vidua*
Snail	*Lymnea* sp.
Sowbug[a]	*Asellus brevicaudis*
Stonefly nymph	*Nemoura* sp.
Stonefly[a]	*Pteronarcella badia*
Stonefly[a]	*Pteronarcys californica*
Water flea	*Daphnia* sp.
Fish	
Black bullhead	*Ictalurus melas*
Bluegill	*Lepomis macrochirus*
Brook trout	*Salvelinus fontinalis*
Brown trout	*Salmo trutta*
Carp	*Cyprinus carpio*
Chain pickerel	*Esox niger*
Channel catfish	*Ictalurnu punctatus*
Chinook salmon	*Oncorhynchus tshawytscha*
Coho salmon	*Oncorhynchus kisutch*
Cutthroat trout	*Salmo clarki*
Dolly Varden trout	*Salvelinus malma*
Fathead minnow	*Pimephales promelas*
Flagfish	*Jordanella floridae*
Gizzard shad	*Dorosoma cepedianum*
Golden shiner	*Notemigonus crysoleucas*
Goldfish	*Carrasius auratus*
Grass carp	*Ctenopharyngodon idella*
Green sunfish	*Lepomis cyanellus*
Lake chubsucker	*Erimyzon sucetta*
Lake trout	*Salvelinus namaycush*
Largemouth bass	*Micropterus salmoides*
Long-nosed killifish	*Fundulus similis*

Table 8-35 (continued)

Species' common and scientific names

Common Name	Scientific Name
Mosquitofish	Gambusia affinis
Northern hogsucker	Hypentelium nigricans
Pink salmon	Oncorhynchus gorbuscha
Pugnose minnow	Notropis emiliae
Rainbow trout	Salmo gairdneri
Sheepshead minnow	Cyprinodon variegatus
Smallmouth bass	Micropterus dolomieui

[a] No other common name available

GLOSSARY

ADI--See acceptable daily intake.

a.e.--See acid equivalent.

a.i.--See active ingredient.

Absorption--The taking up of liquids by solids or the passage of a substance into the tissues of an organism as the result of several processes; that is, diffusion, filtration, or osmosis.

Acceptable daily intake (ADI)--The maximum dose of a substance that is anticipated to be without lifetime risk to humans when taken daily.

Acetylcholine--A chemical involved in transmission (carrying) of nerve impulses across junctions in the nervous system.

Acid equivalent (a.e.)--The amount of active ingredient expressed in terms of the parent acid.

Acre--43,460 ft^2. An area of land about 209 feet long by 209 feet wide.

Active ingredient (a.i.)--The effective part of a pesticide formulation or the actual amount of the technical material present in the formulation.

Actual dosage--The amount of active ingredient (not formulated product) that is applied to an area or other target.

Acute poisoning--Severe poisoning which occurs after one exposure to a pesticide.

Acute toxicity--The potential of a compound to cause injury or illness when given in a single dose or in multiple doses over a period of 24 hours or less. The quality or potential of a substance to cause injury or illness shortly after exposure to a relatively large dose. For aquatic studies, the period of exposure is 96 hours.

Additive--See adjuvant.

Adenoma--An abnormal growth of glandular tissue.

Adjuvant (additive)--Something added to the pesticide mixture to help the active ingredient do a better job. Examples: wetting agent, spreader, adhesive, emulsifying agent, penetrant.

Adsorption--Adhesion of substances to the surfaces of solids or liquids. For example, the attraction of ions of compounds to the surfaces of solids.

Aerosol--Suspension of finely divided particles or droplets in air.

Aliphatic materials--Chemically, those that have an open-chain molecular structure. As herbicides, they are less toxic to plants than aromatic compounds.

Ames assay--A type of short-term test using bacteria in laboratory cultures to assess the mutagenic potential of a substance.

Amine--Any of a group of organic compounds of nitrogen, such as ethylamine, $C_2H_5NH_2$, that may be considered ammonia derivatives in which one or more hydrogen atoms have been replaced by a hydrocarbon radical.

Aromatic oils and solvents--Chemically, those that have unsaturated molecular structure. As herbicides, they are generally more toxic to plants than aliphatic materials.

Assay--A test or measurement used to evaluate a characteristic of a chemical. See bioassay.

BLM--U.S. Department of Agriculture; Bureau of Land Management.

BPA--U.S. Department of Energy; Bonneville Power Administration.

Bioaccumulation--The process of a plant or animal selectively taking in or storing a persistent substance. Over a period of time, a higher concentration of the substance is found in the organism than in the organism's environment.

Bioactivation--A process whereby a plant takes in an apparently harmless chemical, which yields toxic breakdown products when metabolized by the plant.

Bioassay--A method for quantitatively determining the concentration of a substance by its effect on the growth of a suitable animal, plant, or microorganism under controlled conditions.

Bole--A tree stem thick enough to yield saw timber, veneer logs, or large poles.

Boom (herbicide spray)--A tubular metal device that conducts an herbicide mixture from a tank to a series of spray nozzles. It may be mounted beneath a helicopter or behind a tractor.

Broadcast application--Uniform distribution of an herbicide over an entire area.

Broad spectrum pesticides--General-purpose pesticides with a wide range of uses. They are effective when several different pests are a problem to control.

Brown and burn--A method of site preparation in which brush is sprayed with herbicide and, after it has dried out, a controlled fire is set to dispose of the woody material.

CFR--See Code of Federal Regulations.

Cambium--The layer of cells under tree bark that lies between the xylem and phloem and gives rise to secondary growth.

Cancer potency--A measure of the relative ability to cause cancer.

Carcinogen--A substance capable of inciting cancer.

Carcinogenic--Producing or inciting cancer.

Carcinogenicity--Tendency of a substance to cause cancer.

Carcinoma--A malignant or cancerous tumor.

Carrier--The liquid or solid material added to a chemical compound to facilitate its application in the field.

Chemical degradation--The breakdown of a chemical substance into simpler components through chemical reactions.

Chemically inactive--Will not easily react with any other chemical or object. Examples: talc and clay.

Chemical reaction--When two or more substances are combined and as a result undergo a complete change to make new substances or materials.

Chromosome--Microscopic structures within the cell that are composed of DNA and contain the genes (hereditary determiners).

Chronic (effects or toxicity)--Having poisonous or deleterious effects from prolonged exposure or repeated administration of a chemical.

Chronic poisoning--Poisoning which occurs as a result of small, repeated doses of pesticide over a long period of time.

Chronic toxicity--The effects of a series of small doses of a substance applied over a long period that may be related to changes in appetite, growth, metabolism, reproduction, and life span.

Code of Federal Regulations (CFR)--The Code of Federal Regulations is a codification of the general and permanent rules published in the Federal Register by the executive departments and agencies of the Federal Government. The Code is divided into 50 titles that represent broad areas subject to Federal regulations. Each title is divided into chapters, which usually bear the name of the issuing agency. Each chapter is further subdivided into parts covering specific regulatory areas.

The Code of Federal Regulations is kept up to date by the individual issues of the Federal Register. These two publications must be used together to determine the latest version of any given rule.

Cohort study--An epidemiology study where the individuals in the study have one or more common statistical factors (such as age or class membership).

Commercial forest land--Forest land capable of bearing merchantable timber, currently or prospectively accessible, and not withdrawn from such use.

Compatible pesticides--Compounds or formulations that can be mixed and applied together without undesirably altering their separate effects.

Concentration--The amount of active ingredient or acid equivalent in a given volume of liquid or in a given weight of dry material.

Conifer--An order of the Gymnospermae, comprising a wide range of trees, mostly evergreens that bear cones and have needle-shaped or scalelike leaves; timber commercially identified as softwood.

Conjunctivitis--Inflammation of the mucous membrane that lines the inner surface of the eyelids.

Contact herbicide--One that kills primarily by contact with plant tissue rather than as a result of translocation. Toxic upon contact with target or nontarget species.

Cornea--The transparent anterior portion of the outer coat of the vertebrate eye covering the iris and the pupil.

Critical habitat--The specific areas within the geographical area occupied by the species, at the time it is listed in accordance with the Endangered Species Act, on which are found those physical or biological features that are essential to the conservation of the species and that may require special management considerations or protection. Also included are specific areas, outside the geographical area occupied by the species at the time it is listed, which the Secretary determines are essential for the conservation of the species.

Cytogenic--Refers to the structure or function of chromosomes within cells.

DEA--U.S. Department of Justice; Drug Enforcement Agency.

DNA--See deoxyribonucleic acid.

Degrade--To decompose or break up.

Degree of exposure--The amount or extent to which a person has been in contact with a toxic pesticide.

Deoxyribonucleic acid (DNA)--Any of various nucleic acids that are the molecular basis of heredity in many organisms.

Deposit--The pesticide on the leaves or skin or other surface immediately after pesticide application.

Dermal exposure--The portion of a toxic substance that an organism receives as a result of the substance coming into contact with the organism's body surface.

Dermal toxicity--How poisonous a pesticide is to an animal when absorbed through the skin.

Dermatitis--Inflammation of the skin.

Desiccant--An herbicide whose mode of action is through the drying of plant tissues.

Desorption--The removal of ions or compounds attached to the surfaces of particles of soil or organic matter.

Detergent--A chemical (not soap) having the ability to remove soil or grime. Household detergents can be used as surfactants in herbicide sprays.

Diluent--Any liquid or solid material that dilutes an active ingredient in the preparation of a formulation.

Dislodgeable residue--A pesticide residue that can be removed from surfaces, such as foliage, by physical contact.

Disposal--The act or process of discarding or throwing away a pesticide.

Dominant lethal assay--A test to detect a mutation of a dominant gene that may be fatal to the next generation. Usually a male rodent is exposed to a chemical substance and later sequentially mated with two female animals. The females are sacrificed, and the number and status of the fetuses is recorded.

Dormant--Not actively growing.

Dormant spray--Pesticide application made before trees and other plant life begin to leaf out in the spring.

Dosage rate--Quantity of a toxicant applied per unit area. Usually expressed as oz or lbs active ingredient per acre.

Dose--The amount of chemical administered or received by an organism, generally at a given point in time.

Drift--That portion of a sprayed chemical that is moved by wind off a target site.

Duff--The layer of fresh to slightly decomposed organic matter and the less decomposed humus on a forest floor.

Dyspnea--Labored or difficult breathing, sometimes accompanied by pain. Normal when due to vigorous work or athletic activity.

EA--See environmental assessment.

EC_{50}--See median effective concentration.

EEC--Estimated environmental concentration.

EPA--U.S. Environmental Protection Agency.

EPA registration number--A number assigned by EPA to a product when it is registered that must appear on all labels for that product.

Edema--An excessive accumulation of fluid in the cells, tissue spaces, or body cavities caused by a disturbance in the fluid exchange mechanism. Also known as dropsy.

Endangered species--Any species in danger of extinction throughout all or a significant portion of its range that has been designated in the *Federal Register* as an endangered species.

Environmental assessment (EA)--A concise public document that briefly provides sufficient evidence and analysis for determining whether to prepare an Environmental Impact Statement or to return a finding of no significant impact, aids an agency's compliance with NEPA when no Environmental Impact Statement is necessary, or facilitates preparation of a statement when one is necessary.

Environmental fate--The transport, accumulation, and disappearance of an herbicide in the environment.

Environmental impact statement (EIS)--A formal document to be filed with the Environmental Protection Agency that considers significant environmental impacts expected from implementation of a major Federal action.

Ephemeral stream--A stream that flows only in direct response to precipitation and whose channel is above the water table at all times.

Epidemiology--A science that deals with the incidence, distribution, and control of disease in a population.

Ester--A compound formed by the reaction of an acid and an alcohol, generally accompanied by the elimination of water.

Evapotranspiration--The process that returns soil moisture to the atmosphere, including evaporation and plant transpiration (uptake of soil water through roots and loss of water through leaves or needles).

Exposure--The amount of contact with a pesticide.

Exposure analysis--The estimation of the amount of chemicals that organisms receive during the application of pesticides.

FAO--Food and Agricultural Organization (United Nations).

FDA--U.S. Food and Drug Administration.

FIFRA--See Federal Insecticide, Fungicide, and Rodenticide Act.

FSM--See Forest Service Manual.

FWS--Fish and Wildlife Service.

Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)--An act administered by EPA which requires that extensive toxicological studies be conducted on a pesticide in order to assess its potential hazard to humans and the environment.

Federal Register--A daily Federal publication that publishes regulations and legal notices that have been issued by Federal agencies.

Fetotoxic--Capable of producing adverse effects in a developing fetus.

Foliar-acting herbicide--An herbicide that causes localized injury to plant tissue where contact occurs.

Forest Service Manual (FSM)--An internal set of operating directives that governs Forest Service activities.

Formulation--The form in which a pesticide is packaged or prepared for use. A chemical mixture that includes a certain percentage of active ingredient (technical chemical) with an inert carrier.

Fuel--Any substance or composite mixture that can ignite and burn.

Gavage--Feeding by way of a tube inserted into the stomach.

Gene--The basic unit of heredity. Each gene occupies a specific place (locus) on a chromosome.

Genotoxic--Harmful to genetic material (DNA).

Germ cell--A functional sex cell that combines with the opposite sex cell for fertilization. Examples: sperm, egg.

Girdling--Making continuous incisions around a living stem through at least both bark and cambium, generally resulting in the death of the tree.

Granivorous--Feeding on grains and seeds.

Granular products--Formulations in which the chemical is impregnated on or in vermiculite, attaclay, or other suitable carriers and then formed into granules or pellets.

Ground water--Water residing in the interstices of soil and rock below the ground surface.

HDT--Highest dose tested.

HEW--U.S. Department of Health, Education and Welfare (obsolete Departmental name--replaced primarily by HHS and the Department of Education).

HHS--U.S. Department of Health and Human Services.

Habitat--The physical and biological environment of a plant or animal where all essentials for its development and existence are present.

Half-life--The time required for half the amount of a substance (such as an herbicide) in or introduced into a living system to be eliminated, whether by excretion, metabolic decomposition, or other natural process.

Hazard--The risk of danger; the chance that danger or harm will come to the applicator, bystanders, consumers, livestock, wildlife, or crops, etc.

Hazard analysis--The determination of whether a particular chemical is or is not causally linked to particular harmful effects.

Hectare (ha)--10,000 square meters, or approximately 2.47 acres.

Hematology--The science concerned with blood and the blood-forming tissues.

Herbaceous--A plant that does not develop persistent woody tissue above the ground.

Herbicide--A chemical used to control, suppress, or kill plants, or to severely interrupt their normal growth processes.

Herbivore--An animal that exclusively eats plants.

Heritable--Capable of being inherited or of passing to others by inheritance.

Histology--The study of the microscopic structure of tissue.

Histopathology--Study of tissue changes characteristic of disease.

Hydrolysis--Decomposition or alteration of a chemical substance by water.

Hyperplasia--An excessive proliferation of normal cells in the tissue of an organ.

Hypertrophy--An increase in size of an organ or structure that does not involve tumor formation.

Hypohatchet--A tool used to inject herbicide into a tree trunk or woody stem.

IC_{50}--See median immobilization concentration.

Inactive--Will not react chemically with anything; not involved in the pesticide action.

Incompatible--Chemicals that cannot be mixed or used together.

Inert ingredients--All ingredients in a formulated pesticide product that are not classified as active ingredients. Note that inert as used here is a defined usage; many inert products are biologically active chemicals.

Infiltration--The downward entry of water into the soil.

Ingredient statement--The part of the label on a pesticide container that gives the name and amount of each pesticide chemical and the amount of inactive material in the mixture.

Inhalation--To take air into the lungs, to breath in.

Inhalation toxicity--How poisonous a pesticide is to man or an animal when breathed in through the lungs.

Inject--To force a pesticide chemical into a plant, animal, building, or the soil.

Insectivorous--Referring to an animal that eats insects; in common usage, incudes animals that eat insects and sometimes other selected invertebrates.

Intermittent stream--A stream that flows only at certain times of the year when it receives water from springs or from some surface source, such as melting snow.

Interval--The time period between two pesticide applications or between the last pesticide application and harvest.

Intraperitoneal--Related to a structure or process occurring within the peritoneum, a membranous lining of the body cavity.

Intravenous--Within or into a vein.

In vitro--Pertaining to a test that is conducted outside the living body and in an artificial environment such as a test tube or petri dish.

In vivo--Pertaining to a test that is performed within the living body of the organism.

Kilogram (kg)--One thousand grams, or approximately 2.2 pounds.

l--See liter.

LC_{50}--See median lethal concentration.

LD_{50}--See median lethal dose.

LDT--Lowest dose tested.

LEL--Lowest dose level at which toxic effects are observed.

LOEL--See lowest-observed-effect level.

Label--All printed material on or attached to a pesticide container as required by law.

Leach--Usually refers to the movement of chemicals through soil by water; may also refer to the movement of herbicides out of leaves, stems, or roots into the air or soil.

Lethal--Deadly toxic, that is, causing death of target or nontarget species.

Leukemia--A chronic or acute disease characterized by unrestrained growth of leukocytes (white blood cells).

Lowest-observed-effect level (LOEL)--The lowest concentration of a substance that causes any effect in the test organisms.

Lymphoma--A general term for growth of new tissue in the lymphatic system.

MATC--See maximum acceptable toxicant concentration.

mg--See milligram.

mg/kg--Milligrams per kilogram. Used to designate the amount of chemical received per kilogram of body weight of test organisms. 1 mg/kg = 1 ppm. 1 mg = 0.000035 ounce. 1 kg = 2.2 pounds.

mg/kg/day--Milligrams per kilogram of body weight per day.

mg/l--Milligrams per liter of solution.

ml--See milliliter.

MOS--See margin of safety.

Margin of safety (MOS)--The ratio between the animal no-observed-effect level (NOEL) and the estimated human dose. The larger the MOS, the smaller the estimated human dose and the lower the risk to human health. In this risk assessment, if the exposure exceeds the NOEL, then the MOS is expressed as the negative ratio of the exposure to the NOEL.

Maximum acceptable toxicant concentration (MATC)--The hypothetical toxic threshold concentration of a toxicant in water bounded by the highest tested concentration that has no significant adverse effect and the lowest concentration having a significant effect.

Median effective concentration (EC_{50})--The concentration of a chemical at which some effect is observed for 50% of the test organisms. Often used where mortality (as an LC_{50}) is difficult to observe. (The IC_{50} is a specific example of an EC_{50}.)

Median immobilization concentration (IC_{50})--Concentration at which 50 percent of tested aquatic organisms will be immobilized. Used primarily for microorganisms for which it is difficult or impossible to determine whether individual organisms are alive or dead.

Median lethal concentration (LC_{50})--The concentration of a chemical at which 50 percent of the test animals will be killed. It is usually used in testing of fish or other aquatic animals.

Median lethal dose (LD_{50})--The milligram of toxicant per kilogram of animal body weight (mg/kg) lethal to 50 percent of the test animals to which it is administered under the conditions of the experiment.

Metabolic activation--The process of running a mutagenic assay in an environment containing a microsome fraction (centrifugal fraction containing metabolic enzymes).

Metabolism--The chemical changes in living cells by which energy is provided for vital processes and new material is assimilated.

Metabolite--A product of the chemical changes in living cells that provides energy and assimilates new material.

Microbial degradation--The breakdown of a chemical substance into simpler components by bacteria.

Microgram (ug)--One-millionth of a gram.

Milligram (mg)--One-thousandth of a gram.

Mitigate--To cause to become less harsh or harmful.

Mitigation--Actions to avoid, minimize, reduce, eliminate, or rectify the impacts of a management practice.

Mobility--The capability of an herbicide to be moved easily within soil, vertically or laterally, with the normal movement of water.

Mutagen--A substance that tends to increase the frequency or extent of genetic mutations.

Mutagenic--Causing changes in genetic material.

Mutagenicity--The capacity of a substance to cause changes in genetic material.

Mutation--A change in a gene potentially capable of being transmitted to offspring.

NAS--National Academy of Science.

NEPA--See National Environmental Policy Act.

NEPA process--All measures necessary for compliance with the requirements of Section 2 and Title I of NEPA.

NHL--See non-Hodgkin's lymphoma.

NOEL--The no-observed-effect level. In a series of dose levels tested, it is the highest level at which no effect is observed; that is, safe in the species tested.

Necrosis--Death of a cell or group of cells as a result of injury, disease, or other pathologic state.

Negligible residue--A tolerance which is set on a food or feed crop that will have a very small amount of pesticide at harvest as a result of indirect contact with the chemical.

Neuropathy--Any disease affecting neurons, the fundamental functional unit of nervous tissues.

Neurotoxic--Toxic to nerves or nervous tissue.

Nominal concentration--The amount of a substance applied to a surface as opposed to the amount that penetrates that surface to form a solution.

Nonaccumulative--Will not build up in an animal's body or in the environment.

Non-Hodgkin's lymphoma--A new growth of tissue in the lymphatic system that is not considered to be Hodgkin's disease.

Nonpersistent--Lasts only a short time (a few weeks or less) after being applied; breaks down rapidly in the environment.

Nonselective pesticide--A pesticide chemical that will control a wide range of pests.

Nontarget--Any plant, animal, or other organism that a pesticide application is not aimed at, but that may accidentally be injured by the chemical.

Nontarget vegetation--Vegetation that is neither expected nor planned to be affected by herbicide treatment.

Nonvolatile--A pesticide chemical that does not evaporate (turn into a gas or vapor) at normal temperatures.

No-observed-effect level (NOEL)--In a series of dose levels tested, it is the highest level at which no effect is observed.

Noxious weed--A plant regulated or identified by law as being undesirable, troublesome, and difficult to control.

Omnivorous--Eating both animal and vegetable substances.

Oncogenic--Capable of producing or inducing tumors, either benign (noncancerous) or malignant (cancerous), in animals.

Oncology--The branch of medicine that studies tumors.

One-hit model--An equation used to describe the relationship between dose and the probability of contracting cancer. This equation, used at one time by EPA, predicts the greatest cancer probability at low doses of all commonly used models.

Oral--By gavage or fed in the diet.

Organic material--An accumulation of decayed and resynthesized plant and animal residues with a high capacity for holding water and nutrients.

Ossification--The formation of bone.

PADI--See provisional acceptable daily intake.

ppm--See parts per million.

Parenteral--Injection other than into the intestine.

Parts per million (ppm)--The number of parts of the substance in question mixed per million parts of a carrier material. (1 ounce of salt in 62,500 pounds of sugar). One ppm = 1 mg/kg (on a weight basis) = 1 mg/liter (water or air).

Pathology--The study of the nature and cause of disease with respect to functional and structural changes.

Penetrant--A kind of additive or adjuvant that aids the pesticide in getting through the outer surface (leaf, root, skin) and into the plant.

Percolation--The flow of a liquid through a porous substance.

Perennial stream--A stream that flows continuously year round.

Persistence--The resistance of an herbicide to metabolism and environmental degradation and thus an herbicide's retention of its ability to kill plants for prolonged periods.

Pest--An unwanted organism (animal, plant, bacteria, fungus, virus, etc.). See also "weed."

Pesticide--Any substance or mixture of substances intended for controlling insects, rodents, fungi, weeds, or other forms of plant or animal life that are considered to be pests.

Photodecomposition--The breakdown of a substance, especially a chemical compound, into simpler components by the action of radiant energy.

Photolysis--See photodecomposition.

Photooxidation--The process by which exposure to light removes electrons from chemical compounds.

Phytotoxic--Poisonous or harmful to plants.

Piscivorous--Habitually feeding on fish.

Poison--Any chemical or agent that can cause illness or death when eaten, absorbed through the skin, inhaled, or otherwise absorbed by humans, animals, or plants. Note that a substance is a poison or not with respect to specific organisms. Animals may safely eat many things that are "poisonous" to humans.

Preemergent--Applied prior to emergence of the specified weed or planted crop.

Provisional Acceptable Daily Intake (PADI)--An interim value for the ADI of a chemical, pending new data.

Rangeland--Any area on which the vegetation consists of native or introduced grasses, legumes, grasslike plants, forbs, or shrubs, and that is developed for range (grazing) use. Also counted as rangeland are native pastures or meadows that are occasionally cut or mechanically harvested and are grazed by livestock.

Raptors--Birds of prey, such as owls, hawks, or eagles.

Recessive lethal test--A test to detect a mutation of a recessive gene that may be fatal to the next generation.

Recovery plan--An approved Fish & Wildlife Service plan that addresses recovery objectives for a plant or animal species listed as threatened or endangered.

Reentry--The return of a worker to an area that has recently been treated with a pesticide.

Residue--The quantity of an herbicide or its metabolites remaining in or on soil, water, plants, animals, or surfaces.

Residue level--Amount of pesticide that may remain on a crop after harvesting.

Resorption--Act of removal by adsorption.

Riparian areas--Geographically delineated areas, with distinctive resource values and characteristics, that are comprised of the aquatic and riparian ecosystems, floodplains, and wetlands. They include all areas within a horizontal distance of 100 feet from the edge of perennial streams or other water bodies.

Risk--The probability that a substance will produce harm under specified conditions.

Risk analysis--The description of the nature and often the magnitude of risk to organisms, including attendant uncertainty.

Rotation--The number of years required to establish and grow a timber crop to a specified condition of maturity. The rotation includes a period for harvesting and stand re-establishment, usually 5 years.

Runoff--That part of precipitation, as well as any other flow contributions, that appears in surface streams, either perennially or intermittently.

STS--See soft tissue sarcoma.

Safety factor--A factor conventionally used to extrapolate human tolerances for chemical agents from no-observed-effect levels in animal test data.

Sarcoma--Cancer arising from underlying tissue: muscle, bone, and other connective tissue.

Scientific name--A scientific name made up of the genus and species. Sometimes the variety or subspecies is included. This name is more reliable and more universal than common names. The names are based on Latin or Greek.

Sediment--Organic matter or soil that settles to the bottom of a liquid.

Sedimentation--The process or action of depositing sediment.

Selective pesticide, specific pesticide--A pesticide that will control only a few pest species and is not as poisonous to other plants and animals.

Sensitive species--Those species that have appeared in the Federal Register as proposed for classification for official listing as endangered or threatened species or that are on an official State list or are recognized by the Regional Forester to need special management to prevent them from becoming endangered or threatened.

Shock--The severe reaction of the human body to a serious injury. It can result in death if not treated, even if the injury itself would not.

Shrub--A plant with persistent woody stems and relatively low growth form; usually produces several basal shoots as opposed to a single bole; differs from a tree by its low stature and nonarborescent form.

Signal word--Word that must appear on pesticide labels to show how toxic the pesticide is. The signal words used are "Danger-Poison," "Warning," or "Caution."

Sister chromatid exchange assay--Mutation assay designed to evaluate an alteration in the normal exchange of genetic material.

Slash--Woody debris left after logging, pruning, thinning, or brush cutting. It includes logs, chunks, bark, branches, stumps, and broken small trees or brush.

Soft tissue sarcoma (STS)--Cancer arising from soft tissue (nonarticulate tissue).

Soil profile--A vertical section of soil that shows all horizons and parent material.

Solvent--A liquid, such as water, oil, or kerosene, used to dissolve other materials, such as herbicides.

Sorption--The process of taking up or holding by either absorption or adsorption.

Species (plural: species)--A morphologically, genetically, and ecologically defined biological entity to which a binomial and authority is given; for example, Potamogeton filiformis Pers., the slender-leaf Potamogeton.

Spreader-sticker--A surfactant closely related to wetting agents that facilitates spreading and increases sticking of an herbicide on vegetation.

Stand--An aggregation of trees or other growth occupying a specific area and sufficiently uniform in species composition, age, arrangement, and other conditions to be distinguishable from the forest, other growth, or other land cover on adjoining areas.

Subchronic--The effects observed from doses that are of intermediate duration, usually 3 months.

Subcutaneous--Beneath the skin, or to be introduced beneath the skin.

Succession--The gradual supplanting of one community of plants by another.

Surface water--Rivers, lakes, ponds, streams, and so forth, that are located above ground.

Surfactant--A material that improves the emulsifying, dispersing, spreading, wetting, or other surface-modifying properties of liquids.

Susceptible--Can be killed or injured by the pesticide at the rate used.

Suspended sediment--Sediment suspended in a fluid by the upward components of turbulent currents or by colloidal suspension.

Symptom--A warning that something is wrong. An outward signal of a disease or poisoning in a plant, animal, or human.

Synergism--The harmonious action of two agents, producing an effect that neither could produce alone or an effect that is greater than the total effects of each agent operating by itself.

Systemic herbicide--An herbicide that is moved within the plant. In a more restricted sense, refers to herbicides that are applied to foliage and move downward through living tissue to underground parts.

Systemic toxicity--Effects produced as a result of the distribution of a poison or foreign substance from the point of exposure to a distant site within the body.

TLV--See threshold limit value.

Target--The area, buildings, plants, animals, or pests intended to be treated with a pesticide application.

Technical material or pesticide--The pesticide as it is first manufactured by the company before formulation. It is usually almost pure.

Teratogen--A substance tending to cause developmental malformations in unborn human or animal offspring. Teratogenicity is the capacity of a substance to cause anatomical, physiological, or behavioral defects in animals exposed during embryonic development.

Teratogenic--Capable of producing or inciting the development of malformations in an embyro.

Teratogenisis--The development of abnormal structures in an embryo.

Teratology--The study of malformations in organisms.

Test animals--Laboratory animals, usually rats, fish, birds, mice, or rabbits, used to determine the toxicity and hazards of different pesticides.

Threatened species--Any plant or animal species that is likely to become an endangered species within the foreseeable future in all or a significant portion of its range. The species are designated in the Federal Register as threatened species.

Threshold--A dose or exposure below which there is no apparent or measurable adverse effect.

Threshold limit value (TLV)--The concentration of an airborne constituent to which workers may be exposed repeatedly, day by day, without adverse effect.

Tolerance--The amount of a pesticide that can remain on any food (plant or animal) that is to be eaten by livestock or humans. The tolerance is set by the EPA.

Tolerant--Not susceptible to (injured by) a pesticide application.

Toxic--Poisonous, but not necessarily fatal.

Toxicant--A poison.

Toxicity--A characteristic of a substance that makes it poisonous.

Toxicology--The science dealing with the study of the adverse biological effects of chemicals.

Trade name--A brand name. The name given to a pesticide by a manufacturing company to identify it as their product.

Translocated herbicide--One that is moved within the plant from the point of entry.

Translocation--The transfer of substances from one location to another in the plant body.

Transpiration--The process by which plants take up moisture from the soil through their root systems and give off moisture to the air through their leaves (needles).

Transport--Carry from one place to another--usually in a car or truck.

Treated area--A building, field, forest, garden, or other place where a pesticide is applied.

Tumor--A new growth of tissue that forms an abnormal mass and performs no physiologic function. It usually develops independently of and unrestrained by the normal principles of biological growth.

ug--See microgram.

USDA--U.S. Department of Agriculture.

USDA-FS--U.S. Department of Agriculture, Forest Service.

Vapor pressure--The pressure at which a chemical compound will evaporate.

Volatile--A compound is volatile when it evaporates or vaporizes (changes from a liquid to a gas) at ordinary temperatures on exposure to air.

Volatility--The quality of evaporating readily at normal temperatures and pressures.

Volatilization--The vaporizing or evaporating of a chemical substance.

WHO--World Health Organization.

Water table--The upper limit of the part of the soil or underlying rock material that is wholly saturated with water.

Weed--A plant growing where it is not desired.

REFERENCES CITED

REFERENCES FOR SECTION 1

National Research Council. 1983. *Risk Assessment in the Federal Government: Managing the Process.* National Academy Press, Washington, DC.

National Research Council. 1986. *Drinking Water and Health, Volume 6.* R.D. Thomas, ed. National Academy Press, Washington, DC.

U.S. Department of Agriculture, Forest Service. 1984. *Pesticide Background Statements. Volume 1, Herbicides.* Agricultural Handbook 633. Washington, DC.

U.S. Department of Agriculture, Forest Service. 1986. *Pesticide Background Statements. Supplement to Volume 1, Herbicides.* Washington, DC.

U.S. Environmental Protection Agency. 1986. Hazard Evaluation Division. Standard evaluation procedure, ecological risk assessment. Prepared by Urban and Cook. Office of Pesticide Programs, Washington, DC.

Wilson, R. and E.A.C. Crouch. 1987. Risk assessment and comparisons: An introduction. *Science* 236:267-270.

NO REFERENCES FOR SECTION 2

REFERENCES FOR SECTION 3

American Cyanamid Company. 1985a. Arsenal herbicide for brush control and forest management. Agricultural Research Division. Princeton, NJ. 12 pp.

American Cyanamid Company. 1985b. Arsenal herbicide for industrial weed control. Agricultural Research Division. Princeton, NJ. 12 pp.

American Cyanamid Company. 1986. Summary of toxicology data on imazapyr and Arsenal. Agricultural Research Division. Princeton, NJ. 6 pp.

American Cyanamid Company. 1987. Correspondence of Simon K. Hall to Dr. Philip J. Sczerzenie of LABAT-ANDERSON Inc. concerning status of chronic toxicity studies on Arsenal. February 9, 1987.

American Petroleum Institute. 1983a. 28-day dermal toxicity study in the rabbit, hydrodesulfurized kerosene sample 81-07. American Petroleum Institute Medical Research Publication No. 30-32297.

American Petroleum Institute. 1983b. Carcinogenic potential of key petroleum products. American Petroleum Institute Medical Research Publication 30-31646.

Anderson, J.W. 1975. Laboratory studies on the effects of oil on marine organisms: An overview. American Petroleum Institute Publication No. 4249.

Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The acute toxicology of selected petroleum hydrocarbons. In: MacFarland, H.N., C.E. Holdsworth, J.A. MacGregor, R.W. Call, and M.L. Kane (eds.). Proceedings of the Symposium . . . The Toxicology of Petroleum Hydrocarbons, pp. 1-12. American Petroleum Institute, Washington, DC.

Beliles, R.P. and F.J. Mecler. 1982. Inhalation teratology of jet fuel A, fuel oil, and petroleum naphtha in rats. In: MacFarland, H.N., C.E. Holdsworth, J.A. MacGregor, R.W. Call, and M.L. Kane (eds.). Proceedings of the Symposium . . . The Toxicology of Petroleum Hydrocarbons, pp. 233-238. American Petroleum Institute, Washington, DC.

Bingham E., R.P. Trosset, and D. Warshawsky. 1979. Carcinogenic potential of petroleum hydrocarbons. Journal of Environmental Pathology and Toxicology 3:483-563.

Biodynamics Inc. Undated. Interim report: A chronic dietary toxicity and oncogenicity study with AC 243,997 in rats. Project No. 84-2862. East Millstone, NJ.

Blackburn, G.R., R.A. Dietch, C.A. Schreiner, and C.R. Mackerer. 1984. Correlation of in vitro mutagenicity with dermal carcinogenicity for complex, petroleum-derived mixtures. Environmental Mutagenesis 6:454.

BLM. See U.S. Department of the Interior, Bureau of Land Management.

Brusick, D. 1980. Principles of Genetic Toxicology. Plenum Press, New York.

California Department of Food and Agriculture (CDFA). 1986. Summary of toxicological data for fosamine.

Canadian Centre for Toxicology. 1987. Expert Panel Report on Carcinogenicity of 2,4-D, March 23, 1987. Guelph, Ontario, Canada. 67 pp.

Cantor, K.P. and A. Blair. 1986. Agricultural chemicals, drinking water, and public health: An epidemiologic overview. National Cancer Institute, Bethesda, MD.

Colton, T. 1986. Herbicide exposure and cancer. Journal of American Medical Association 256(9):1,176-1,178.

Conaway C.C., C.A. Schreiner, and S.T. Cragg. 1982. Mutagenicity evaluation of petroleum hydrocarbons. In: MacFarland, H.N., C.E. Holdsworth, J.A. MacGregor, R.W. Call, and M.L. Kane (eds.). Proceedings of the Symposium . . . The Toxicology of Petroleum Hydrocarbons, pp. 128-138. American Petroleum Institute, Washington, DC.

Crump, K.S. 1983. Statement as expert witness in the case of Northwest Coalition for Alternatives to Pesticides (NCAP) et al. versus J.R. Block et al. U.S. District Court for the District of Oregon. Civil No. 83-6273-E.

DOE. See U.S. Department of Energy.

Doull, J., C.D. Klassen, and M.O. Amdur. 1980. Cassarett and Doull's Toxicology, 2nd ed. MacMillan, New York.

Dow Chemical Company. 1985. Correspondence of Richard J. Nolan, Mammalian and Environmental Toxicology Research Laboratory, Dow Chemical Company to Larry Gross, U.S. Forest Service, regarding triclopyr toxicology data. October 22, 1985. Midland, MI.

Dow Chemical Company. 1986a. Material data sheet for Garlon 3A and Garlon 4 herbicides. Agricultural Products Department. Midland, MI.

Dow Chemical Company. 1987a. Supplemental information concerning triclopyr and picloram (with additional 2,4-D comments) for use in development of a vegetation management environmental impact statement for the Southern Region. Submitted to U.S. Forest Service and LABAT-ANDERSON, Incorporated. March 1987.

Dow Chemical Company. 1987b. Correspondence of Alice H. Morgan, Agricultural Products Department, Dow Chemical to Allan J. West, U.S. Forest Service, regarding inert ingredients of Dow pesticidal products. March 10, 1987.

Draize, J.H., G. Woodard, and H.O. Calvery. 1944. Methods for the study of irritation and toxicity of substances applied topically to the skin and mucous membranes. Journal of Pharmological Experimental Theory 82:377-390.

DuPont Chemical Company. 1983a. Technical data sheet for fosamine amonium. Biochemicals Department. Wilmington, DE. 5 pp.

DuPont Chemical Company. 1983b. Technical data sheet for sulfometuron methyl. Agricultural Chemicals Department. Wilmington, DE. 6 pp.

DuPont Chemical Company. 1984a. Technical data sheet for hexazinone. Agricultural Chemicals Department. Wilmington, DE. 7 pp.

DuPont Chemical Company. 1984b. Material safety data sheets for Pronone 5G and 10G herbicides. Agricultural Products Department. Wilmington, DE.

DuPont Chemical Company. 1985. Material safety data sheet for Velpar L herbicide. Agricultural Products Department. Wilmington, DE.

DuPont Chemical Company. 1986. Material safety data sheet for Oust herbicide. Agricultural Products Department. Wilmington, DE.

Elanco Products Company. 1989. Letter to the Forest Service in response to the DEIS Vegetation Management in the Appalachian Mountains. Division of Eli Lilly and Company. Greenfield, Indiana.

Elegbede, J.A., C.E. Elson, M.A. Tanner, A. Qureshi, and M.N. Gould. 1986a. Regression of rat primary mammary tumors following dietary d-limonene. Journal of the National Cancer Institute 76(2):323-325.

Elegbede, J.A., C.E. Elson, M.A. Tanner, and M.N. Gould. 1986b. Inhibition of DNMA-induced mammary cancer by the monoterpene d-limonene. Carcinogenesis 5(5):661-664.

Environ Corporation. 1985. Elements of Toxicology and Chemical Risk Assessment. Washington, DC.

EPA. See U.S. Environmental Protection Agency.

Eriksson, M.L., L. Hardell, N.O. Berg, T. Moller, and O. Axelson. 1981. Soft tissue sarcomas and exposure to chemical substances: Case reference study. British Journal of Industrial Medicine 38:27-33.

Fisher, H.L., B. Most, and L.L. Hall. 1985. Dermal absorption of pesticides calculated by deconvolution. Journal of Applied Toxicology 5(3):163-177.

Food and Agriculture Organization/World Health Organization. 1986. Pesticide residues in food--1986. Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and a WHO Expert Group on Pesticide Residues, Rome, 29 September-8 October 1986. FAO Plant Production and Protection Paper 77.

Furia, T.E. and N. Bellanca. 1975. Fenaroli's Handbook of Flavor Ingredients. The Chemical Rubber Company, Cleveland, OH.

Goldstein, N.P., P.H. Jones, and J.R. Brown. 1959. Peripheral Neuropathy after exposure to an ester of dichlorophenoxyacetic acid. Journal of American Medical Association 130:1306-1309.

Green, L.M. 1986. Mortality analysis of Ontario Hydro forestry tradesmen cohort 1950-1982: Executive summary. Health Services Department, Health and Safety Division, Ontario Hydro, Toronto, Canada.

Grissom, R.E., Jr., C. Brownie, and F.E. Guthrie. 1985. Dermal absorption of pesticides in mice. Pesticide Biochemistry and Physiology 24:119-123.

Hansen, W.H., M.L. Quaife, R.T. Haberman, and O.G. Fitzhugh. 1971. Chronic toxicity of 2,4-dichlorophenoxyacetic acid in rats and dogs. Toxicology and Pharmacology 20:122-129.

Hardell, L. and A. Sandstrom. 1979. Case-control study: Soft tissue sarcomas and exposure to phenoxyacteic acids or chlorophenols. British Journal of Cancer 39:7711-7717.

Hardell, L., M. Eriksson, P. Lenner, and E. Lundgren. 1981. Malignant lymphoma and exposure to chemicals especially organic solvents, chlorophenols and phenoxy acids: A case-control study. *British Journal of Cancer* 43:169-176.

Hayes, A.W. 1982. *Principles and Methods of Toxicology*. Raven Press, New York. 359 pp.

Hazardous Substance Data Bank. 1987a (March). Kerosene, HSN 632. National Library of Medicine, Bethesda, MD.

Hazardous Substance Data Bank. 1987b (February). Limonene, HSN 1809. National Library of Medicine, Bethesda, MD.

Hazelton Laboratories. 1986. Pathology summary carcinogenicity in mice with 2,4-dichlorothenoxactic acid, unscheduled deaths and terminal sacrifice. Unpublished report. Project #2184-101. Vienna, VA.

Hoar, S.K., A. Blair, F.F. Holmes, C.D. Boysen, R.J. Robel, R. Hoover, and J.F. Fraumeni. 1986. Agricultural herbicide use and risk of lymphoma and soft-tissue sarcoma. *Journal of American Medical Association* 256(9):1,141-1,147.

HSDB. See Hazardous Substance Data Bank.

JLB International Chemical Inc. 1983. Cide-Kick: Acute inhalation toxicity study report. Vero Beach, FL.

John-Greene, J.A., J.H. Ouellette, T.K. Jeffries, K.A. Johnson, and K.S. Rao. 1985. Teratological evaluation of picloram potassium salts in rabbits. *Food Chemistry and Toxicology* 23(8):753-756.

Labat-Anderson, Inc. 1986. Sulfometuron methyl background statement. Arlington, VA.

Labat-Anderson, Inc. 1988a. Imazapyr background statement. Arlington, VA.

Labat-Anderson, Inc. 1988b. Light fuel oil background statement. Arlington, VA.

Lavy, T.L. and J.D. Mattice. 1986. Progress in pesticide exposure studies and future concerns. *Toxicology Letters* 33:61-71.

Loomis, T.A. 1978. *Essentials of Toxicology*. Lea and Febiger, Philadelphia, PA.

MacMahon, B. 1986. A review of the Hoar et al. study on agricultural herbicide use and risk of lymphoma and soft tissue sarcoma. Report prepared for the Environmental Protection Agency. Harvard School of Public Health, Cambridge, MA.

Mattsson, J.L., R.R. Albee, K.A. Johnson, and J.F. Quast. 1986a. Neurotoxicologic examination of rats dermally exposed to 2,4-D amine for three weeks. *Neurobehavioral Toxicology and Teratology* 8:255-263.

Mattsson, J.L., K.A. Johnson, and R.R. Albee. 1986b. Lack of neuropathologic consequences of repeated dermal exposure to 2,4-dichlorophenoxyacetic acid in rats. Fundamental and Applied Toxicology 6:175-181.

Mecler, F.J. and R.P. Beliles. 1979. Teratology study in rats. Diesel fuel final report. American Petroleum Institute Medical Research Publication No. 27-32174.

Monsanto Company. 1983. Material safety data sheet for Rodeo herbicide. St. Louis, MO.

Monsanto Company. 1986. N-Nitrosoglyphosate information sheet. October 1986. St. Louis, MO. 4 pp.

Mullison, W.R. 1985. A toxicological and environmental review of picloram. The Dow Chemical Company, Midland, MI. 92 pp.

National Cancer Institute. 1978. Bioassay of picloram for possible carcinogenicity. Carcinogenesis Technical Report Series 23. Bethesda, MD.

National Research Council (NRC). 1986. Drinking Water and Health. Volume 6. R.D. Thomas, ed. National Academy Press, Washington, D.C.

NCI. See National Cancer Institute.

Newton, M. and F.N. Dost. 1981. Environmental Effects of Vegetation Management Practices on DNF Forest Lands. Prepared for State of Washington, Department of Natural Resources.

NRC. See National Research Council.

Parodi, S., S. DeFlora, M. Carvanna, A. Pino, L. Robbiano, C. Bennicelli, and G. Brambilla. 1981. DNA-damaging activity in vivo and bacterial mutagenicity of sixteen hydrazine derivatives as related quantatatively to their carcinogenicity. Cancer Research 41:1469-1482.

Parodi, S., M. Taningher, P. Boero, and L. Santi. 1982. Quantitative correlations amongst alkaline DNA fragmentation, DNA covalent binding, mutagenicity in the Ames test and carcinogenicity, for 21 compounds. Mutatation Research 93(1):1-24.

Parodi, S., M. Taningher, P. Ruso, M. Pala, D. Vecchio, G. Fassina, and L. Santi. 1983a. Quantitative predictivity of the transformation in vitro assay compared with the Ames test. Journal of Toxicology and Environmental Health 12(4-6):483-510.

Parodi, S., A. Zunino, L. Ottaggio, M. DeFerrari, and L. Santi. 1983b. Quantitative correlation between carcinogenicity and sister chromatid exchange induction in vivo for a group of 11 N-nitroso derivatives. Journal of Toxicology and Environmental Health 11(3):337-346.

Pearce, N.E., A.J. Smith, J.K. Howard, R.A. Sheppard, H.J. Giles, and C.A. Teague. 1986. Non-Hodgkin's lymphoma and exposure to phenoxy-herbicides, chlorophenols, fencing work, and meat works employment: A case control study. British Journal of Industrial Medicine 43:75-83.

Pogodina, O.N., M.P. Svetlova, N.V. Tomilin, G.B. Pliss, and V.V. Khudolei. 1984. Correlation of mutagenic and carcinogenic activities of certain aromatic amines. Eksp. Onkol. 6(4):23-25.

SCM Organic Chemicals. 1981. Material Safety Data Sheet. Jacksonville, FL.

Smith, A.H., N.E. Pearce, D.O. Fisher, H.J. Giles, C.A. Teague, and J.K. Howard. 1984. Soft tissue sarcoma and exposure to phenoxy-herbicides and chlorophenols in New Zealand. Journal of the National Cancer Institute 73:1111-1117.

Starek, A. and M. Kaminski. 1981. Comparative studies on toxicity of various dielectrics, petroleum derivatives, used in electroerosion technology. IV. Morphological and cytoenzymatic changes in the lungs and acid-based imbalance in rats chronically exposed to petroleum hydrocarbons. Med. Pr. 32(2):69-81.

Starek, A. and M. Kaminski. 1982. Comparative studies on the toxicity of various dielectrics--petroleum derivatives used in the electroerosion technic. V. Functional, morphological and cytoenzmatic changes in the kidneys of rats chronically exposed to petroleum hydrocarbons. Med. Pr. 33(1-3):1-14.

Thomas, R.D., ed. 1986. Drinking Water and Health. Volume 6. National Research Council. National Academy Press, Washington, DC.

USDA. See U.S. Department of Agriculture.

U.S. Department of Agriculture, Forest Service. 1984. Pesticide Background Statements, Volume I: Herbicides. Agriculture Handbook No. 633. U.S. Government Printing Office, Washington, DC.

U.S. Department of Agriculture, Forest Service. 1986. Pesticide Background Statements, Volume I: Herbicides (Supplement). Agriculture Handbook No. 633. U.S. Government Printing Office, Washington, DC.

U.S. Department of Energy, Bonneville Power Administration. 1983. Final Environmental Impact Statement: Transmission Facilities Vegetation Management Program. DOE/EIS-0097-F. U.S. Government Printing Office, Washington, DC. 464 pp. and appendixes.

U.S. Department of Health and Human Services, Public Health Service. 1985. Fourth annual report on carcinogens. Summary 1985. NTP 85-002. Washington, DC.

U.S. Department of the Interior, Bureau of Land Management. 1985. Northwest Area Noxious Weed Control Program Environmental Impact Statement. Oregon State Office, Portland, OR.

U.S. Environmental Protection Agency. 1974. Toxicity categories. Federal Register. October 16, 1974:39(201).

U.S. Environmental Protection Agency. 1978. Proposed guidelines for registering pesticides in the U.S. hazard evaluation: humans and domestic animals. Federal Register, August 22, 1978:37335-37403.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1982b. Review of toxicology studies submitted by Union Carbide on 2-(2,4 dichlorophenoxy)-propionic acid. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1982d. Guidance for the reregistration of pesticide products containing hexazinone. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1983. Guidance for the reregistration of pesticide products containing dicamba. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1983a. Dicamba science chapters. Washington, D.C.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984a. Summary of results of studies submitted in support of the registration of 2,4-DP. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984b. Summary of results of studies submitted in support of the registration of dicamba. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984c. Summary of results of studies submitted in support of the registration of picloram. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984d. Summary of results of studies submitted in support of the registration of sulfometuron methyl. Washington, DC.

U.S. Environmental Protection Agency. 1984e. Summary of results of studies submitted in support of the registration of tebuthiuron. Office of Pesticides and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984f. Picloram science chapters. Washington, DC.

U.S. Environmental Protection Agency. 1985. Code of Federal Regulations (40 CFR). Part 180 50(84):184-185.

U.S. Environmental Protection Agency. 1985a. Review of preliminary draft supplement to methods of managing competing vegetation: A programmatic environmental impact statement. Letter to James L. Stewart, U.S. Forest Service. November 22. Washington, DC.

U.S. Environmental Protection Agency. 1985b. Pesticide fact sheet for imazapyr. September 5, 1985. Washington, DC. 5 pp.

U.S. Environmental Protection Agency. 1985c. Status report on the toxicological studies under review, or expected to be reviewed for the herbicides paraquat, glyphosate, and 2,4-D. Memorandum to Charles Sherman, Drug Enforcement Administration. October 3. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1985d. Memorandum to Office of Federal Affairs, EPA, on 2,4-D, glyphosate, and paraquat studies. August 7. Washington, DC.

U.S. Environmental Protection Agency. 1986a. Chemical information fact sheet for 2,4-D. August 1986. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986b. Summary of results of studies submitted in support of the registration of 2,4-D. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986c. Summary of results of studies submitted in support of the registration of glyphosate. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986d. Summary of results of studies submitted in support of the registration of hexazinone. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986e. Summary of results of studies submitted in support of the registration of tebuthiuron. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986f. Summary of results of studies submitted in support of the registration of triclopyr. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986g. Summary of results of studies submitted in support of the registration of dicamba. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986h. Letter to John Neisess, U.S. Forest Service, regarding the NOEL for dicamba. Washington, DC. April 4, 1986.

U.S. Environmental Protection Agency. 1986i. Correspondence to Sandoz Crop Protection Corporation concerning EPA's conclusions/comments on the dicamba rat feeding and oncogenicity study. Robert J. Taylor, Registration Division. November 1986. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1986j. Guidance for the reregistration of pesticide products containing glyphosate as the active ingredient. Washington, DC.

U.S. Environmental Protection Agency. 1986k. Guidelines for mutagenicity risk assessment. Federal Register 51(185):34006-34012. September 24, 1986.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986L. Review of draft human health risk assessment for the use of pesticides in the nurseries of the U.S. Forest Service by Toxicology Branch, Hazard Evaluation Division. Washington, DC.

U.S. Environmental Protection Agency, Office of External Affairs. 1986m. Memorandum of carcinogenic risk assessments of 2,4-D. Washington, DC.

U.S. Environmental Protection Agency. 1986n. Health Effects Assessment for Benzene. NTIS PB No. 86 134483/AS. Environmental Criteria and Assessment Office. Cincinnati, OH.

U.S. Environmental Protection Agency. 1986o. Health Effects Assessment for Benzene. NTIS PB No. 86 134335/AS. Environmental Criteria and Assessment Office. Cincinnati, OH.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1987a. Summary of results of studies submitted in support of the registration of fosamine. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1987b. Correspondence of Robert Taylor, EPA, to Kevin Cannon, Monsanto Company, regarding review of glyphosate dog study. June 9, 1987. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1987c. Guidance for the reregistration of pesticide products containing tebuthiuron as the active ingredient. July 1987. Washington, DC.

U.S. Environmental Protection Agency. 1987d. Correspondence to Guy Baier on inert ingredients. February 19, 1987. Washington, DC.

U.S. Environmental Protection Agency, Office of Drinking Water. 1987e. Dicamba Health Advisory (Draft). Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1987f. Guidance for the reregistration of pesticide products containing tebuthiuron as the active ingredient. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Subtances. 1988. Summary of results of studies submitted in support of the registration of picloram. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1988b. ADI Tracking Report, October 20, 1988. Washington, DC.

Van Duuren, B.L. and B.M. Goldschmidt. 1976. Cocarcinogenic and tumor-promoting agents in tobacco carcinogenesis. Journal of the National Cancer Institute 56(6):1237-1242.

Vertac Chemical Corporation. 1977. Material safety data sheet for Weedar 64. Memphis, TN.

Vertac Chemical Corporation. 1982. Material safety data sheet for Esteron 99. Memphis, TN.

Walstad, J.D. and F.N. Dost. 1984. The health risks of herbicides in forestry: A review of the scientific record. Publication 10. Forest Research Station, Oregon State University.

Wiklund, K. and L.E. Holm. 1986a. Soft-tissue sarcoma risk in Swedish agricultural and forestry workers. Journal of the National Cancer Institute 76:229-234.

Wiklund, K. and L.E. Holm. 1986b. Trends in cancer risks among Swedish agricultural workers. Journal of the National Cancer Institute 77:657-664.

Wolfsdorf, J. and K. Paed. 1976. Kerosene intoxications: An experimental approach to the etiology of the CNS manifestations in primates. Journal of Pediatrics 88(6):1037-1040.

Woods, J.A., L. Polissar, R.K. Severson, L.S. Heuser, and B.G. Kulander. 1987. Soft tissue sarcoma and non-Hodgkin's Lymphoma in relation to phenoxy herbicide and chlorinated phenol exposure in western Washington. Journal of the National Cancer Institute (in press).

World Health Organization. 1984. Environmental Health Criteria 29: 2,4-Dichlorophenoxyacetic Acids (2,4-D). Geneva, Switzerland. 151 pp.

REFERENCES FOR SECTION 4

Alton, J.D. and J.F. Stritzke. 1973. Degradation of dicamba, picloram, and four phenoxy herbicides in soils. Weed Science 21(6):556-560.

Anderson, H.A. 1982. Aids to determining fuel models for estimating fire behavior. Gen. Tech. Rep. INT-122. USDA, Forest Service Intermountain Forest and Range Experiment Station. Ogden, UT. 22 pp.

Ateshian, J.K.H. 1974. Estimation of rainfall erosion index. J. Irrig. and Drainage Division ASCE 100 (IR3):293-307.

Bovey, R.W., F.S. Davis, and M.G. Merkle. 1967. Distribution of picloram in huisache after foliar and soil applications. Weeds 15:245-249.

Brady, N.C. 1974. *The Nature and Properties of Soils*. 8th ed. Macmillan, New York.

Carsel, R.F., C.N. Smith, L.A. Mulkey, J.D. Dean, and P.P. Jowise. 1984. *Users Manual for the Pesticide Root Zone Model (PRZM) Release 1*. U.S. Environmental Protection Agency, Environmental Research Laboratory. Athens, GA. EPA/600/3-84/109.

Chang, S.S. and J.F. Stritzke. 1977. Sorption, movement, and dissipation of tebuthiuron in soils. *Weed Science* 25:184-187.

Dean, J.D., P.P. Jowise, and A.S. Donigian, Jr. 1984. *Leaching Evaluation of Agricultural Chemicals (LEACH) Handbook*. Prepared for the U.S. Environmental Protection Agency, Environmental Research Laboratory, Athens, GA. Anderson-Nichols and Co., Inc. EPA-600/3-84-068.

Dow Chemical U.S.A. 1987c. Correspondence of John L. Troth, Agricultural Products Department, Dow Chemical U.S.A., to Mike Srago, U.S. Forest Service regarding triclopyr dermal absorption. December 23, 1987.

Draper, W.M. and J.C. Street. 1982. Application exposure to 2,4-D, dicamba, and a dicamba isomer. *J. Environ. Sci. Health* B17(4):321-339.

Fears, R.D. and S.A. McMaster. Undated. Evaluation of helicopter mounted application systems for potential spray drift. Dow Chemical USA, Midland, MI.

Feldman, R.J. and H.I. Maibach. 1974. Percutaneous penetration of some pesticides and herbicides in man. *Toxicol. Appl. Pharmacol*. 28:126-132.

Franklin, C.A., R. Grover, J.W. Markham, A.E. Smith, and K. Yashida. 1982. Effect of various factors on workers involved in aerial spraying of herbicides. *Transactions of the Canadian Journal of Industrial Hygiene* 43:97-117.

Ghassemi, M., L. Fargo, Page Painter, Pam Painter, S. Quinlivan, R. Scofield, and A. Takata. 1981. *Environmental Fate and Impacts of Major Forest Use Pesticides*. Prepared for the U.S. Environmental Protection Agency, Offices of Pesticides and Toxic Substances. TRW Environmental Division, Redondo Beach, CA.

Grissom, R.E., Jr., C. Brownie, and F.E. Guthrie. 1985. Dermal absorption of pesticides in mice. *Pesticide Biochemistry and Physiology* 24:119-123.

Grover, R. 1973. The adsorptive behavior of acid and ester forms on 2,4-D on soils. *Weed Research* 13:51-58.

Haith, D.A. 1980. A mathematical model for estimating pesticide losses in runoff. *J. Environ. Qual*. 9:428-433.

Hance, R.J. 1976. Adsorption of glyphosate by soils. *Pesticide Science* 7:363-366.

Harvey, J. Jr., J.J Dulka, and J.J. Anderson. 1985. Properties of sulfometuron methyl affecting its environmental fate: aqueous hydrolysis and photolysis, mobility and adsorption on soils, and bioaccumulation potential. J. Agric. Food Chem. 33:590-596.

Hoerger, F. and E.E. Kenaga. 1972. Pesticide residues on plants: Corelation of representative data as a basis for estimation of their magnitude in the environment. In F. Coulston (ed.), EQS Environmental Quality and Safety. Vol. 1. Global Aspects of Toxicology and Technology as Applied to the Environment. Academic Press, New York.

Konikow, L.F. and J.D. Bredehoeft. 1986. Computer model of two-dimensional salute transport and dispersion in ground water. Chapter C2, Book 7, Techniques of Water Resources Investigations of the United States Geological Survey. (Updated 1986).

Lavy, T.L., J.D. Mattice, and L.A. Norris. 1984. Exposure of forestry applicators using formulations containing 2,4-D, dichlorprop, or picloram in non-aerial applications. Project completion report to U.S. Department of Agriculture, Forest Service. Corvallis, OR. 214 pp.

Lavy, T.L., J.D. Walstad, R.R. Flynn, and J.D. Mattice. 1982. (2,4-Dichlorophenoxy) acetic acid exposure received by aerial application crews during forest spray operations. J. Agric. Food Chem. 30:375-381.

Lavy, T.L., J.S. Shepard, and J.D. Mattice. 1980. Exposure measurements of applicators spraying (2,4,5-Trichlorophenoxy) acetic acid in the forest. J. Agric. Food Chem. 28:626-630.

Libich, S., J.C. To, R. Frank, and G.J. Sirons. 1984. Occupational exposure of herbicide applicators to herbicides used along electric power transmission line right-of-way. Am. Ind. Hyg. Assoc. J. 45(1):56-62.

Makary, M.H., J.C. Street, and R.P. Sharma. 1986. Pharmicokinetics of dicamba isomers applied dermally to rats. Pesticide Biochemistry and Physiology 25:258-263.

Mayeux, H.S., Jr., C.W. Richardson, R.W. Bovey, E. Burnett, M.G. Merkle, and R.E. Meyer. 1984. Dissipation of picloram in storm runnoff. J. Environ. Qual. 13(1):44-49.

Michael, J.L. 1986. Fate of Arsenal in forest watersheds after aerial applications for forest weed control. U.S. Forest Service. Final Report Summary. 4110, FS-SO-4105-1.20 (Problem 1). Auburn, AL.

Michael, J.L. and D.G. Neary. 1987. Movement of sulfometuron methyl in forest watersheds after aerial application of Oust (R) for herbaceous weed control. U.S. Forest Service Service. Final Report Summary. 4110 (4300)(1342) FS-SO-4105-1.19 (Problem 1) FS-SE-4151-26. Gainsville, FL.

Mullison, W.R. 1985. A toxicological environmental review of picloram. *Proceedings of the 38th Annual Meeting of the Western Society of Weed Science*. March 1985. Phoenix, AZ.

Mullison, W.R. 1986. An interim report summarizing 2,4-D toxicological research sponsored by the industry task force on 2,4-D research data and a brief review of 2,4-D environmental effects. Industry Task Force on 2,4-D Research Data, Washington, DC.

Nash, R.G., P.C. Kearney, J.C. Maitler, C.R. Sell, and S.N. Fertig. 1982. Agricultural applicators exposure to 2,4-dichlorophenoxyacetic acid. In Pesticide Residue and Exposure, p. 119-132, J.R. Plimmer (ed.). American Chemical Society Symposium Series 182. Washington, DC.

Neary, D.G. 1985. Fate of pesticides in Florida's forests: An overview of potential impacts on water quality. *Soil and Crop Science Society of Florida Proceedings*. Volume 44:18-24.

Neary, D.G., P.B. Bush, J.E. Douglas, and R.L. Todd. 1985. Picloram movement in an appalachian hardwood forest watershed. *Journal of Environmental Quality* 14(4):585-592.

Neary, D.G. 1987. Personnel communication with D.G. Neary in regard to Triclopyr field data.

Neary, D.G., P.B. Bush, and J.E. Douglass. 1983. Off-site movement of hexazinone in stormflow and baseflow from forest watersheds. *Weed Science* 31:543-551.

Newton, M. and F.N. Dost. 1981. *Environmental Effects of Vegetation Management Practices on DNF Forest Lands*. Prepared for State of Washington, Department of Natural Resources.

Newton, M. and L.A. Norris. 1981. Potential exposure of humans to 2,4,5-T and TCDD in the Oregon Coast ranges. *Fundamental and Applied Toxicology* 1:339-346.

Newton, M., A. Allen, D.E. White, B.R. Kelpsas, and F. Roberts. 1982. Deposits and degradation of herbicides in sclerophyll brushfield foliage. Western Society of Weed Science. Denver, Colorado.

Nolan, R.J., N.L. Freshour, P.E. Kastl, and J.H. Saunders. 1983. Picloram: Pharmacokinetics in Human Volunteers. Toxicology and Applied Pharmacology. Research Laboratory, Health and Environmental Science, Dow Chemical USA, Midland, MI.

Rao, P.S.C. and J.M. Davidson. 1980. Estimation of pesticide retention and transformation parameters required in nonpoint source pollution models. In M.R. Overcash, and J.M. Davidson (editors), *Environmental Impact of Nonpoint Source Pollution*. Ann Arbor Science Publishers, Inc., Ann Arbor, MI.

Rhodes, R.C. 1980. Soil studies with ^{14}C-Labeled Hexazinone. J. Agric. Food Chem. 28:311-315.

Sprankle, P., W.F. Meggitt, and D. Penner. 1975. Adsorption, mobility, and microbial degradation of glyphosate in the soil. Weed Science 23(3):229-234.

Stewart, D.K.R. and S.O. Gaul. 1977. Persistence of 2,4-D, 2,4,5-T and Dicamba in a dykeland soil. Bulletin of Environmental Contamination and Toxicology. 18(2):210-218.

Travis, C.C. 1978. Mathematical description of adsorption and transport of reactive solutes in soil: A review of selected literature. Oak Ridge National Laboratory (ORNL-5403), Oak Ridge, TN. 67 pp.

USDA. See U.S. Department of Agriculture.

U.S. Department of Agriculture, Forest Service. 1972. Forest land erosion and sediment evaluation. Forest Service Handbook FSH 3509.21 NA.

U.S. Department of Agriculture, Forest Service. 1976. Southern forestry smoke management guidebook. Gen. Tech. Rep. SE-10. Southeastern Forest Experiment Station, Asheville, NC. 140 pp.

U.S. Department of Agriculture, Forest Service. 1984. Pesticide Background Statements, Volume I: Herbicides. Agriculture Handbook No. 633. U.S. Government Printing Office, Washington, DC.

U.S. Department of Agriculture, Forest Service. 1986. Pesticide Background Statements, Vol. I: Herbicides--Supplement: Tebuthiuron. Argiculture Handbook No. 633. U.S. Government Printing Office, Washington, D.C.

U.S. Department of Agriculture, Soil Conservation Service. 1972. SCS National Engineering Handbook, Section 4, Hydrology. U.S. Government Printing Office, Washington, DC.

U.S. Environmental Protection Agency. 1973. Methods of identifying and evaluating the nature and extent of nonpoint sources of pollutants. Office of Air and Water Programs. U.S. Government Printing Office, Washington, DC.

U.S. Environmental Protection Agency. 1986a. State Program Briefs: Pesticides in Ground Water. Office of Ground-Water Protection, Washington, DC.

U.S. Environmental Protection Agency. 1986b. Pesticides in Ground Water: Background Document. Office of Ground-Water Protection, Washington, DC.

Weeks, A. 1985. Unpublished data, National Institutes of Health.

Wester, R.C. and H.I. Maibach. 1985. In vivo percutaneous absorption and decontamination of pesticides in humans. *Journal of Toxicology and Environmental Health* 16:25-37.

Wischmeier, W.H. and D.D. Smith. 1978. *Predicting Rainfall Erosion Losses--A Guide to Conservation Planning*. U.S. Department of Agriculture, Agriculture Handbook No. 537. U.S. Government Printing Office, Washington, DC.

Yates, W.E., N.B. Akesson, and D.E. Bayer. 1978. Drift of glyphosate sprays applied with aerial and ground equipment. *Weed Science* 26(6):597-604.

Yates, W.E., R.E. Cowden, and N.B. Akesson. 1985. Atomization of herbicide simulants with hollow cone and Raindrop nozzles. Report No. FPM 85-1. USDA Forest Service, Forest Pest Management, Davis, CA.

Yi-lan, W., J. Xi-peng, J. Xue-zhi, L. Hui-fen, and L. Feng. 1984. Percutaneous absorption of radioactively labelled pesticides. *Chinese Medical Journal* 97(6):443-446.

REFERENCES FOR SECTION 5

ACGIH (American Conference of Governmental Industrial Hygienists). 1984. TLV's (threshold limit values) for chemical substance in the work environment adopted by ACGIH for 1984-85. Cincinnati, OH. 116 pp.

American Cyanamid. 1985. Arsenal herbicide for brush control and forest management. Agricultural Research Division. Princeton, NJ. 12 pp.

Brusick, D. 1980. *Principles of Genetic Toxicology*. Plenum Press, New York. 279 pp.

Bush, P.B., D.G. Neary, C.K. McMahon, and J.W. Taylor. 1987. Suitability of hardwoods treated with phenoxy and pyridine herbicides for use as firewood. *Arch. Environ. Contam. Toxicol*. 16:333-341.

Calabrese, E.J. 1978. *Pollutants and High Risk Groups*. John Wiley and Sons, New York.

Calabrese, E.J. 1984. *Ecogenetics*. John Wiley and Sons, New York.

Calabrese, E.J. and M.W. Dorsey. 1984. *Healthy Living in an Unhealthy World*. Simon and Schuster, New York. (Paperback, 1985).

Crouch, E.A.C. and R. Wilson. 1982. Risk/Benefit Analysis. Ballinger, Cambridge, MA.

Davies, J.E., V.H. Freed, H.F. Enos, R.C. Duncan, A. Barquet, C. Morgade, L. Peters, and J. Danauskas. 1982. Reduction of pesticide exposure with protective clothing for applicators and mixers. *Journal of Occupational Medicine* 24(6):464-468.

Dost, F.N. 1983. An analysis of human health hazards associated with some herbicides used in forestry. Prepared for the Bureau of Land Management, Oregon State Office, Portland, OR. 39 pp.

Dost, F.N. 1986. An estimate of carcinogenic risk associated with polyaromatic'hydrocarbons in smoke from prescribed burning in forestry. Oregon State University. Corvallis, OR. 26 pp.

Doull, J., C.D. Klassen, and M.O. Andur. 1980. Casarett and Doull's Toxicology, 2nd ed. MacMillan Publishing Co., New York, NY.

Dourson, M.L. and J.J.F. Stara. 1983. Regulatory history and experimental support of uncertainty (safety) factors. Regulatory Toxicol. and Pharmacol. 3:224-238.

Dow Chemical Company. 1987. Supplemental information concerning triclopyr and picloram (with additional 2,4-D comments) for use in development of a vegetation management environmental impact statement for the Southern Region. Submitted to U.S. Forest Service and LABAT-ANDERSON, Incorporated. March 1987.

DuPont De Nemours Co., Inc. 1986. Comment letter on Draft Supplement to the Western Oregon Program - Management of Competing Vegetation dated April 25, 1985 by R.B. Frigitt, Agricultural Chemicals Department.

DuPont De Nemours Co., Inc. 1987. Product information for Dupont herbicides (krenite, oust, and velpar). 3 pp.

Hansen, W.H., M.L. Quaife, R.T. Haberman, and O.G. Fitzhugh. 1971. Chronic toxicity of 2,4-dichlorophenoxyacetic acid in rats and dogs. Toxicol. and Pharmacol. 20:122-129.

Howe, R.B. and K.S. Crump. 1982. GLOBAL 82: A computer program to extrapolate quantal animal toxicity data to low doses. Prepared for the Office of Carcinogen Standards, OSHA, U.S. Department of Labor, Contract 41USC252C3.

Klaassen, C.D. and J. Doull. 1980. Evaluation of safety: toxicologic evaluation. In Doull, J., C.D. Klaassen, and M.O. Amdur Casarett and Doull's Toxicology: the Basic Science of Poisons, chap. 2. MacMillan, New York. 778 pp.

Kociba, R.J. and W.R. Mullison. 1985. Toxicological interactions with ag chemicals. Farm Supplier August.

Kolmodin-Hedman, B., S. Hoglund, and M. Akerblom. 1983. Studies on phenoxy acid herbicides. I. Field study: occupational exposure to phenoxy acid herbicides (MCPA, Dichlorprop, Mecoprop and 2,4-D) in agriculture. Arch. Toxicol. 54:257-265.

Lavy, T.L., J.D. Mattice, and L.A. Norris. 1984. Exposure of forestry applicators using formulations containing 2,4-D, dichlorprop, or picloram in non-aerial applications. Project completion report to U.S. Department of Agriculture, Forest Service. Corvallis, OR. 214 pp.

Lavy, T.L., J.D. Walstad, R.R. Flynn, and J.D. Mattice. 1982. (2,4-Dichlorophenoxy) acetic acid exposore received by aerial application crews during forest spray operations. *Journal of Agriculture and Food Chemistry* 30:375-381.

Libich, S., J.C. To, R. Frank, and G.J. Sirons. 1984. Occupational exposure of herbicide applicators to herbicides used along electric power transmission line right-of-way. *Am. Ind. Hyg. Assoc. J.* 45(1):56-62.

Mantel, N. and M.A. Schneiderman. 1975. Estimating "safe levels," a hazardous undertaking. *Cancer Research* 35:1379-1386.

Marzulli, F.N. and H.I. Maibach. 1983. *Dermatotoxicology*. (2nd ed.). Hemisphere Publishing Company, Washington, DC. 693 pp.

McMahon, C.K., H.B. Clements, P.B. Bush, D.G. Neary, and J.W. Taylor. 1985. Pesticides released from burning treated wood. *In Proceedings, Eighth Conference on Fire and Forest Meteorology*, pp. 145-152.

Mullison, W.R. 1985. A toxicological and environmental review of picloram. The Dow Chemical Company, Midland, MI. 92 pp.

National Academy of Science (NAS). 1977. *Drinking Water and Health*. Volume 1. Washington, DC.

Office of Science and Technology Policy. 1985. Chemical carcinogens: a review of the science and its associated principles. *Federal Register*, March 14, 1985: 10371-10442.

Putnam, A.R., M.D. Willis, L.K. Binning, and P. F. Boldt. 1983. Exposure of pesticide applicators to nitrofen: Influence of formulation, handling systems, and protective garments. *J. Agric. Food Chem.* 31:645-650.

Thomas, R.D. (ed.). 1986. *Drinking Water and Health*. Volume 6. National Academy Press. Washington, DC.

U.S. Department of Agriculture, Forest Service. 1980. Summary of 2,4-D; 2,4-DP; and 2,4,5-T residues found in water during or after operational projects. Unpublished report. Portland, OR.

U.S. Department of Agriculture, Forest Service. 1984. *Pesticide Background Statements, Volume I: Herbicides*. Agriculture Handbook No. 633. U.S. Government Printing Office, Washington, DC.

U.S. Department of Agriculture, Forest Service; Animal and Plant Health Inspection Service. 1985a. Final Environmental Impact Statement as Supplemented: Gypsy Moth Suppression and Eradication Projects. U.S. Government Printing Office, Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984a. Summary of results of studies submitted in support of the registration of amitrole. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984b. Summary of results of studies submitted in support of the registration of asulam. Washington, DC.

U.S. Environmental Protection Agency. 1984c. Summary of results of studies submitted in support of the registration of limonene. Washington, DC.

U.S. Environmental Protection Agency. 1985. (January 4). Preliminary Risk Assessment for Bromacil Based on Haskel Study #893-80 in CD-1 Mice, CAS #111. Memorandum Hazard Evaluation Division, Office of Pesticides and Toxic Substances, Washington, DC.

U.S. Environmental Protection Agency, Office of External Affairs. 1986a. Memorandum of carcinogenic risk assessments of 2,4-D. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986b. Summary of results of studies submitted in support of the registration of 2,4-D. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1986c. Guidance for the reregistration of pesticide products containing glyphosate as the active ingredient. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986d. Summary of results of studies submitted in support of the registration of hexazinone. Washington, DC.

U.S. Environmental Protection Agency. 1986e. Guidelines for the health risk assessment of chemical mixtures. Federal Register 51(185):34014-34025. September 24, 1986.

U.S. Environmental Protection Agency. 1987. Summary of results of studies submitted in support of the registration of fosamine. Washington, DC.

Volk, W.A. and M.F. Wheeler. 1983. Basic Microbiology, 5th Edition. Harper and Row, Publishers, New York, NY.

Weed Science Society of America (WSSA). 1983. Herbicide Handbook. Weed Science Society of America, 5th ed. Champaign, IL.

Waldron, A.C. 1985. Minimizing pesticide exposure risk for the mixer-loader, applicator, and field worker. R.C. Honeycutt, G. Zweig, and N.N. Ragsdale, eds. In: Dermal Exposure Related to Pesticide Use. ACS Symposium Series No. 273. American Chemical Society.

REFERENCES FOR SECTION 6

API. See American Petroleum Institute.

American Cyanamid Company. 1984. Summary of additional wildlife and nontarget insect toxicity data on Arsenal. Agricultural Research Division, Princeton, NJ. 2 pp.

American Cyanamid Company. 1985. Arsenal herbicide for brush control and forest management. Agricultural Research Division, Princeton, NJ. 12 pp.

American Petroleum Institute. 1982. Project No. 1597. *Index and Abstracts for Reports and Other Publications*. Medicine and Biological Science Department. American Petroleum Institute, Washington, DC. 44 pp.

American Petroleum Institute. 1983. *28-Day Dermal Toxicity Study in the Rabbit, Hydrodesulfurized Kerosene Sample 81-07*. American Petroleum Institute Medical Research Publication No. 30-32297.

Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The acute toxicology of selected petroleum hydrocarbons. *In* MacFarland, H.N., C.E. Holdsworth, J.A. MacGregor, R.W. Call, and M.L. Kane (eds.), *Proceedings of the Symposium . . . The Toxicology of Petroleum Hydrocarbons*. American Petroleum Institute, Washington, DC.

Beliles, R.P. and F.J. Mecler. 1982. Inhalation teratology of jet fuel A, fuel oil, and petroleum naphtha in rats. *In* MacFarland, H.N., C.E. Holdsworth, J.A. MacGregor, R.W. Call, and M.L. Kane (eds.), *Proceedings of the Symposium . . . The Toxicology of Petroleum Hydrocarbons*. American Petroleum Institute, Washington, DC.

Burks, S.L. 1982. *Effects of Oil on Aquatic Organisms: A Review of Selected Literature*. Prepared for the American Petroleum Institute, Washington, DC. 69 pp.

Butler, P.A. 1965. Effects of herbicides on estuarine faunci. *Proceedings of the Southern Weed Conference* 18:576-580.

Calabrese, E.J. 1983. *Principles of Animal Extrapolation*. John Wiley and Sons, New York. 603 pp.

Chrzanowski, R.L., J.C.-Y. Han, and C.L. McIntosh. 1979. Metabolism of $[^{14}C]$ fosamine ammonium in the rat. *Journal of Agriculture and Food Chemistry* 27(3):550-554.

Connell, D.W. and G.J. Miller. 1984. *Chemistry and Ecotoxicology of Pollution*. John Wiley and Sons, New York. 444 pp.

DEA. See U.S. Department of Justice, Drug Enforcement Administration.

DOE. See U.S. Department of Energy.

Dow Chemical Company. Undated. Technical information on triclopyr, the active ingredient of Garlon herbicides. Agricultural Productions Department, Midland, MI.

Dow Chemical Company. 1981. Evaluation of Garlon 4 formulation in the aquatic environment. Environmental Sciences Research Laboratory.

Dow Chemical Company. 1985. Correspondence of Richard J. Nolan, Mammalian and Environmental Toxicology Research Laboratory, Dow Chemical Company to Larry Gross, U.S. Forest Service regarding triclopyr toxicology data. October 22, 1985. Midland, MI.

Dow Chemical Company. 1987. Supplemental information concerning triclopyr and picloram (with additional 2,4-D comments) for use in development of a vegetation management environmental impact statement for the Southern Region. Submitted to U.S. Forest Service and LABAT-ANDERSON Incorporated by the Dow Chemical Company. March 1987.

DuPont Chemical Company. 1983a. Technical data sheet for fosamine amonium. Biochemicals Department, Wilmington, DE. 5 pp.

DuPont Chemical Company. 1983b. Technical data sheet for sulfometuron methyl. Agricultural Chemicals Department, Wilmington, DE. 6 pp.

DuPont Chemical Company. 1984. Technical data sheet for hexazinone. Agricultural Chemicals Department, Wilmington, DE. 7 pp.

DuPont Chemical Company. 1986. Technical aspects of DuPont sulfonylurea herbicides for industrial weed control. Agricultural Products Development, Wilmington, DE.

EPA. See U.S. Environmental Protection Agency.

Erne, K. 1974. Phenoxy herbicide residues in Swedish fish and wildlife. In F. Coulston, F. Korte, F. Klein, and I. Rosenblum (eds.), Pesticides Environmental Quality and Safety. Suppl. Vol. III, Georg Thieml Publishers, Stuttgart, West Germany.

Folmar, L.C., H.O. Sanders, and A.M. Julin. 1979. Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates. Archives of Environmental Contamination and Toxicology 8:269-278.

Gersich, F.M., C.G. Mendoza, D.L. Hopkins, and K.M. Bodner. 1984. Acute and chronic toxicity of triclopyr triethylamine salt to Daphnia magna Straus. Bulletin of Environmental Contamination and Toxicology 32:497-502.

Gersich, F.M., D.L. Hopkins, and D.P. Milazzo. 1985. Acute and chronic toxicity of technical picloram (4-amino-3,5,6-trichloropicolinic acid) to Daphnia magna Straus. Bulletin of Environmental Contamination and Toxicology 35:121-126.

Ghassemi, M., L. Fargo, Page Painter, Pam Painter, S. Quinlivan, R. Scofield, and A. Takata. 1981. Environmental Fates and Impacts of Major Forest Use Pesticides. Prepared for the U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. TRW Environmental Division, Redondo Beach, CA.

HSDB. See Hazardous Substances Data Bank.

Halter, M.T. 1980. 2,4-D in the aquatic environment. In Literature Reviews of Four Selected Herbicides: 2,4-D, Dichlobenil, Diquat, and Endothall. Municipality of Metropolitan Seattle, Seattle, WA.

Hazardous Substances Data Bank (HSDB). 1987a (March). Dicamba, HSN 311. National Library of Medicine, Bethesda, MD.

Hazardous Substances Data Bank (HSDB). 1987b (March). Kerosene, HSN 632. National Library of Medicine, Bethesda, MD.

Hazardous Substances Data Bank (HSDB). 1987c (March). Picloram, HSN 1151. National Library of Medicine, Bethesda, MD.

Hoffman, D.J. and P.H. Albers. 1984. Evaluation of potential embryotoxicity and teratogenicity of 42 herbicides, insecticides, and petroleum contaminants to mallard eggs. Archives of Environmental Contamination and Toxicology 13:15-27.

Hooser, S.B., V.R. Beasley, J.I. Everitt. 1986. Effects of an insecticidal dip containing d-limonene in the cat. Journal of the American Veterinary Medical Association 189(8):905-908

Hudson, R.H., R.K. Tucker, and M.A. Haegle. 1984. Handbook of Toxicity of Pesticides to Wildlife. 2nd ed. U.S. Department of the Interior, Fish and Wildlife Service, Washington, DC.

ITII. See International Technical Information Institute.

Inglis, A. and E.L. Davis. 1972. Effects of water hardness on the toxicity of several organic and inorganic herbicides to fish. Technical Paper No. 67. Bureau of Sport Fisheries and Wildlife, Columbia, MO.

International Technical Information Institute (ITII). 1976. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan.

Irvine, A.M., J.C. Forbes, and S.R. Draper. 1977. Effect of 2,4-D on the water-soluble carbohydrate content of ragwort (Senecio jacobaea L.) leaves. Weed Research 17:169-172.

JLB International Chemical, Inc. Undated. CIDE-KICK II technical bulletin. Vero Beach, FL.

JLB International Chemical, Inc. 1983. Cide-Kick: Acute inhalation toxicity study report. Vero Beach, FL.

JLB International Chemical, Inc. 1987. Material safety data sheet for CIDE-KICK. Vero Beach, FL.

Jackson, J.B. 1965. Toxicologic studies of a new herbicide in sheep and cattle. American Journal of Veterinary Research 27(118):821-824.

John-Greene, J.A., J.H. Ouelette, T.K. Jeffries, K.A. Johnson, and K.S. Rao. 1985. Teratological evaluation of picloram potassium salt in rabbits. Journal of Food Chemistry and Toxicology 23(8):753-756.

Johnson, C.R. 1976. Herbicide toxicities in some Australian anurans and the effect of subacute dosages on temperature tolerance. Zoological Journal of the Linnean Society 59:79-83.

Johnson, W.W. and M.T. Finley. 1980. Handbook of acute toxicity of chemicals to fish and aquatic invertebrates. Resource Publication No. 137. U.S. Department of the Interior, Fish and Wildlife Service, Washington, DC.

Kenaga, E.E. 1969. Tordon herbicides--Evaluation of safety to fish and birds. Down to Earth 25(1):5-9.

Kenaga, E.E. 1979. Acute and chronic toxicity of 75 pesticides to various animal species. Down to Earth 35(2):25-31.

Kopischke, E.D. 1972. The effect of 2,4-D and diesel fuel on egg hatchability. Journal of Wildlife Management 36:1353-1356.

Lagier, R.F. et al. 1974. Adjuvants decrease insecticide hazard to honey bees. College of Agriculture Research Center Bulletin No. 801. Washington State University, Pullman, WA.

Lehman, H.J. 1954. Appraisal of the safety of chemicals in food, drugs, and cosmetics. Association of Food and Drug Officials of the United States 18:66.

Linden, E., B.E. Bengtsson, O. Svanberg, and G. Sunstrom. 1979. The acute toxicity of 78 chemicals and pesticide formulations against two brackish water organims, the Bleak (Alburnus alburnus) and the Harpacticoid (Nitocra spinipes). Chemosphere 8(11-12):843-851.

Lutz-Ostertag, Y. 1983. The toxic and teratogenic effects of the ammonium salt of fosamine (a defoliant) on the development of quail and chick embryos. Archives d'Anatomie Microscopique 72(1):77-85.

Lynn, G.E. 1965. A review of toxicological information on Tordon herbicides. Down to Earth 20(4):6-8.

Mayer, F.L. Jr. and M.R. Ellersieck. 1986. Manual of acute toxicity: interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication 160. U.S. Department of the Interior, Fish and Wildlife Service, Washington, DC.

Mayes, M.A. and D.C. Dill. 1984. The acute toxicity of picloram, picloram potassium salt, and picloram triisopropanolamine salt to aquatic organisms. *Environmental Toxicology and Chemistry* 3:263-269.

Mayes, M.A., D.C. Dill, K.M. Bodner, and C.G. Mendoza. 1984. Triclopyr triethylamine salt toxicity to life stages of the fathead minnow (*Pimephales promelas* Rafinesque). *Bulletin of Environmental Contamination and Toxicology* 33:339-347.

Mayes, M.A., D.L. Hopkins, and D.C. Dill. 1987. Toxicity of picloram (4-amino-3,5,6,-trichloropicolinic acid) to life stages of the rainbow trout. *Bulletin of Environmental Contamination and Toxicology* 38:653-660.

McCollister, D.C. and M.L. Leng. 1969. Toxicology of picloram and safety evaluation of Tordon herbicides. *Down to Earth* 25(2):5-10.

Mecler, F.J. and R.P. Beliles. 1979. Teratology study in rats. Diesel fuel final report. American Petroleum Institute Medical Research Publication No. 27-32174.

Melzian, B.D. 1983. Acute toxicity, histopathy, and bioconcentration. retention studies with No. 2 fuel oil and the blue crab, *Callinectes sapidus* Rathbun. *Dissertation Abstracts International B: The Sciences and Engineering* 43(11).

Mitchell, D.G., P.M. Chapman, and T.J. Long. In press. Acute toxicity of the glyphosate herbicides Roundup and Rodeo to rainbow trout, chinook, and coho salmon.

Moffett, J.O., H.L. Morton, and R.L. MacDonald. 1972. Toxicity of some herbicidal sprays to honey bees. *Journal of Economic Entomology* 65(1):32-36.

Monsanto Company. Undated. Toxicity information provided by Tim Long of Monsanto Agricultural Products Division to Paul Mistretta, U.S. Forest Service, 1987.

Monsanto Company. 1982. The health and environmental safety aspects of Roundup herbicide: An overview. Roundup Herbicide Bulletin No. 3. Monsanto Company, St. Louis, MO. 6 pp.

Monsanto Company. 1983. Rodeo herbicide: Toxicological and environmental properties. Bulletin No. 1. Agricultural Products Company, Environmental Practices, St. Louis, MO. 4 pp.

Monsanto Company. 1985a. Material safety data for Roundup herbicide. St. Louis, MO. 6 pp.

Monsanto Company. 1985b. Material Safety Data Sheet for Glyphosate Technical. 6pp.

Mullison, W.R. 1985. A toxicological and environmental review of picloram. The Dow Chemical Company, Midland, MI. 92 pp.

Osweiler, G.D. 1983. Toxicology of triclopyr herbicide to equine. American Association of Veterinary Laboratory Diagnosticians, 20th Annual Proceedings: 193-202.

Palmer, J.S. and R.D. Radeleff. 1969. The toxicity of some organic herbicides to cattle, sheep, and chickens. Production Research Report No. 106. U.S. Department of Agriculture, Agricultural Research Service, Washington, DC. 23 pp.

Pimentel, D. 1971. Ecological effects of pesticides on non-target species. Executive Office of the President. Office of Science and Technology. Washington, DC.

Sanders, H.O. 1969. Toxicity of pesticides to the crustacean Gammarus lacustris. Technical Paper No. 25. U.S. Department of the Interior. Fish and Wildlife Service, Bureau of Sport Fisheries and Wildlife, Washington, DC.

Sanders, H.O. 1970. Toxicities of some herbicides to six species of freshwater crustaceans. Journal of the Water Pollution Control Federation 42(8):1544-1550.

Sanders, H.O. and O.B. Cope. 1968. The relative toxicities of several pesticides to naiads of three species of stoneflies. Limnology and Oceanography 13(1):112-117.

Sheppard, D.C. 1987. The orange peel mystery. Science of Food and Agriculture (March):9-12.

Sprague, J.B. 1985. Factors that modify toxicity. In Fundamentals of aquatic toxicology. Rand, G.M. and S.R. Petrocelli (eds.). Hemisphere Publishing Corporation, New York.

Sullivan, T.P. 1985. Effects of glyphosate on selected species of wildlife. In The Herbicide Glyphosate. Grossbard, E. and D. Atkinson (eds.). Buttersworth, Boston, MA.

Szaro, R.C., P.H. Albers, and N.C. Coon. 1978. Petroleum: Effects on mallard egg hatchability. Journal of Wildlife Management 42:404-406.

Tooby, T.E., J. Lucey, and B. Stott. 1980. The tolerance of grass carp, Ctenopharyngodon idella Val., to aquatic herbicides. Fish Biology 16:591-597.

Tsuda, S. and Y. Okuno. 1985. Solvents and insecticidal efficacy of the aerosol containing tetramethrin and d-phenothrin. Journal of Pesticide Science 10(4):621-628.

U.S. Department of Agriculture (USDA), Forest Service. 1984. Pesticide Background Statements, Volume I: Herbicides. Agriculture Handbook No. 633. U.S. Government Printing Office, Washington, DC.

U.S. Department of Agriculture, Forest Service. 1986. Pesticide Background Statements, Volume I: Herbicides (Supplement). Agriculture Handbook No. 633. U.S. Government Printing Office, Washington, DC.

U.S. Department of Energy, Bonneville Power Administration. 1983. Final Environmental Impact Statement: Transmission Facilities Vegetation Management Program. Appendices DOE/EIS-0097-F. U.S. Government Printing Office, Washington, DC.

U.S. Department of Justice, Drug Enforcement Administration (DEA). 1986. Final Environmental Impact Statement: Cannabis Eradication on Non-Federal and Indian Lands in the Contiguous United States and Hawaii. U.S. Government Printing Office, Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1982. Guidance for the reregistration of pesticide products containing hexazinone. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1983a. Dicamba science chapters. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides Programs. 1983b. Guidance for the reregistration of pesticide products containing dicamba as the active ingredient. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984a. Summary of results of studies submitted in support of the registration of 2,4-DP. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984b. Summary of results of studies submitted in support of the registration of glyphosate. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984c. Summary of results of studies submitted in support of the registration of hexazinone. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984d. Summary of results of studies submitted in support of the registration of limonene. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984e. Picloram science chapters. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1984f. Summary of results of studies submitted in support of the registration of sulfometuron methyl. Washington, DC.

U.S. Environmental Protection Agency. 1985a. Chemical information fact sheet for imazapyr. September 1985. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1985b. Summary of results of studies submitted in support of the registration of triclopyr. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1985c. Hazard Evaluation Division, Standard Evaluation Procedure, acute toxicity test for freshwater fish. Prepared by E. Zucker. EPA-540/9-85-006. Washington, DC. NTIS PB86-129277.

U.S. Environmental Protection Agency. 1986a. Chemical information fact sheet for 2,4-D. August 1986. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides Programs. 1986b. Tox 1-liner for dicamba. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides Programs. 1986c. Tox 1-liner for glyphosate. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1986d. Guidance for the reregistration of pesticide products containing glyphosate as the active ingredient. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1986e. Summary of results of studies submitted in support of the registration of tebuthiuron. Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1986f. Summary of studies submitted in support of registration of triclopyr. Washington, D.C.

U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. 1987. Correspondence of Robert Taylor, EPA, to Kevin Cannon, Monsanto Company, regarding review of glyphosate dog study. June 9, 1987. Washington, DC.

Watkins, C.E. and D.E. Thayer. 1982. Toxicity of four surfactants to bluegill sunfish. Quarterly Report. Center for Aquatic Weeds, University of Florida, Gainesville, FL. USDA/ARS No. 58-7B30-0-177.

Woodward, D.F. 1976. Toxicity of the herbicides dinoseb and picloram to cutthroat (Salmo clarki) and lake trout (Salvelinus namaycush). Journal of the Fisheries Research Board of Canada 33(8):1671-1676.

Woodward, D.F. 1979. Assessing the hazard of picloram to cutthroat trout. Journal of Range Management 32(3):230-232.

REFERENCES FOR SECTION 7

Altman, P.L. and D.S. Dittmer (eds.). 1962. Growth: Including Reproduction and Morphological Development. Fed. American Society of Experimental Biology, Washington, DC. 608 pp.

Auffenberg, W. and J.B. Iverson. 1979. Demography of terrestrial turtles. In Turtles: Perspectives and Research, pp. 541-569.

Banse, K. and S. Mosher. 1980. Adult body mass and annual production/biomass relationships of field populations. Ecological Monographis 50(3):355-379.

Beal, F.E.L. 1911. Food of the woodpeckers of the U.S. U.S. Department of Agriculture Biological Survey Bulletin No. 37.

Bureau of Land Management. 1987. Supplement to the Northwest Area Noxious Weed Control Program Environmental Impact Statement. U.S. Department of the Interior, Washington, DC.

Burt, W.H. and R.P. Grossenheider. 1966. A Field Guide to the Mammals. Houghton-Mifflin, Boston.

Chapman, F.M. 1966. Handbook of Birds of Eastern North America. Dover Publications, New York. 581 pp.

Conant, R. 1958. A Field Guide to Reptiles and Amphibians (of the United States and Canada East of the 100th Meridian). Houghton-Mifflin, Boston. 366 pp.

Damuth, J. 1981. Home range, home range overlap, and species energy use among herbivorous mammals. Zoological Journal of the Linnean Society 15:185-193.

Davis J.R. 1978. Gray Squirrel Management in Alabama. Alabama Department of Conservation and Natural Resources. Special Report No. 7. 29 pp.

Davis, J.R. 1979. The White-Tailed Deer in Alabama. Alabama Department of Conservation and Natural Resources. Special Report No. 8. 60 pp.

Davis, W.B. 1974. The Mammals of Texas. Texas Game and Fish Commission Bulletin.

Dickerson, M.C. 1969. The Frog Book: North American Toads and Frogs. Dover Publications, New York. 253 pp.

Dickson, J.G. 1978. Seasonal bird populations in a south central Louisiana bottomland hardwood forest. Journal of Wildlife Management 42(4):875-883.

Drozdz, A. 1968. Digestibility and assimilation of natural foods in small rodents. Acta Theriologica 13(21):367-389.

Gordon, M.S., G.A. Bartholomew, A.D. Grinnell, C.B. Jorgensen, and F.N. White. 1968. Animal Function: Principles and Adaptations. MacMillan Company, New York.

Hamilton, W.J., Jr. 1941. The food of small forest mammals in eastern United States. Journal of Mammalogy 22:250-263.

Hamilton, W.J., Jr. and J.O. Whitaker, Jr. 1979. Mammals of the Eastern United States. Comstock Publishing Associates, Cornell University Press, Ithaca, NY. 346 pp.

Hoerger, F. and E.E. Kenaga. 1972. Pesticide residues on plants: correlation of representative data as a basis for estimation of their magnitude in the environment. In F. Coulston (ed.). EQS: Environmental Quality and Safety, Volume I. Global Aspects of Toxicology and Technology as Applied to the Environment. Academic Press, New York.

Hutchinson, V.H., W.G. Whitford, and M. Kohl. 1968. Relation of body size to gas exchange in anurans. Physiological Zoology 41:65-85.

Kendeigh, S.C. 1969. Tolerance of cold and Bergmann's rule. The Auk 86:13-25.

Kendeigh, S.C. 1970. Energy requirements for existence in relation to size of bird. The Condor 72:60-65.

Komarek, E.V. and R. Komarek. 1938. Mammals of the Great Smoky Mountains. Bulletin of the Chicago Academy of Sciences 5(6):137-162.

Lasiewski, R.C. and W.A. Calder, Jr. 1971. A preliminary allometric analysis of respiratory variables in resting birds. Respiration Physiology 11:152-166.

Lasiewski, R.C. and W.R. Dawson. 1967. A reexamination of the relation between standard metabolic rate and body weight in birds. The Condor 69:13-23.

Lockie, J.D. 1959. Estimation of the food of foxes. Journal of Wildlife Management 23(2):224-227.

Lowery, G.H., Jr. 1974. The Mammals of Louisiana and Its Adjacent Waters. Louisiana State University Press, Baton Rouge. 565 pp.

Meyers, J.M. and A.S. Johnson. 1978. Bird communities associated with succession and management of loblolly-shortleaf pine forests. In Proceedings of the Workshop: Management of Southern Forests for Nongame Birds, pp. 50-65. U.S. Department of Agriculture-Forest Service General Technical Report SE-14.

Moore, J.A. 1964. Physiology of the Amphibia. Academic Press, New York. 654 pp.

Odum, E.P. 1949. Small mammals of the highlands (North Carolina) plateau. Journal of Mammalogy 30(2):179-192.

Odum, E.P. 1971. Fundamentals of Ecology. W.B. Saunders, Co., Philadelphia. 574 pp.

Odum, E.P., C.E. Connell, and L.B. Davenport. 1962. Population energy flow of three primary consumer components of old-field ecosystems. Ecology 43(1):88-96.

Prickett, T. Undated. Louisiana Bobwhite Basics. Louisiana Department of Wildlife and Fisheries Wildlife Education Bulletin No. 113. 38 pp.

Robbins, C.S., B. Bruun, H.S. Zim, and A. Singer. 1966. A Guide to Field Identification: Birds of North America. Golden Press, New York.

Sargeant, A.B. 1978. Red fox prey demands and implications to prairie duck production. Journal of Wildlife Management 42(3):520-527.

Schmidt, J.L. and D.L. Gilbert. 1978. Big Game of North America. Stackpole, Harrisburg, PA.

Schmidt-Nielsen, K. 1972. How Animals Work. Cambridge University Press.

Schmidt-Neilsen, K. 1975. Animal Physiology. Prentice-Hall, Englewood Cliffs, NJ. 145 pp.

Schwartz, C.W. and E.R. Schwartz. 1959. The Wild Mammals of Missouri. University of Missouri Press and Missouri Conservation Commission.

Scott, V.E., K.E. Evans, D.R. Patton, and C.P. Stone. 1977. Cavity nesting birds of North American forests. U.S. Department of Agriculture Handbook No. 511.

Seehorn, M.E. 1982. Reptiles and amphibians of southeastern National Forests. U.S. Department of Agriculture Forest Service Southern Region. 85 pp.

Seibert, H.C. 1949. Differences between migrant and non-migrant birds in food and water intake at various temperatures and photoperiods. Auk 66:128-153.

Slobodkin, L.B. and S. Richman. 1961. Calories/gm. in species of animals. Nature 4785:299.

Sturkie, P.D. 1965. Avian Physiology. Comstock Publishing Associates, Cornell University Press, Ithaca, NY. 766 pp.

U.S. Drug Enforcement Administration. 1985. Final Environmental Impact Statement on the Eradication of Cannabis on Federal Lands in the Continental United States. U.S. Department of Justice, Washington, DC.

U.S. Drug Enforcement Administration. 1986. Final Environmental Impact Statement on the Eradication of Cannabis on Non-Federal and Indian Lands in the Contiguous United States and Hawaii. U.S. Department of Justice, Washington, DC.

U.S. Environmental Protection Agency. 1984. Ecological effects disciplinary review. Memorandum on Thiram Registration Standard from J.J. Bascietto, Ecological Effects Branch, Hazard Evaluation Division, TS-769c to E. Wilson, PM Team 21, Registration Division, TS-767c.

Welty, J.C. 1962. The Life of Birds. W.B. Saunders, Philadelphia. 546 pp.

Wood, G.W. and L.J. Niles. 1978. Effects of management practices on nongame bird habitat in longleaf-slash pine forests. In Proceedings of the Workshop: Management of Southern Forests for Nongame Birds, pp. 40-49. U.S. Department of Agriculture-Forest Service General Technical Report SE-14.

Zar, J.H. 1968. Standard metabolism comparisons between orders of birds. The Condor 10(3):278.

REFERENCES FOR SECTION 8

American Cyanamid Co. 1986. Summary of environmental fate studies. Agricultural Research Division. Princeton, NJ.

Dennis, D.S., W.H. Gillespie, R.A. Maxey, and R. Shaw. 1977. Accumulation and persistence of picloram (Tordon 10K) in surface water and bottom sediments in West Virginia. Arch. Environ. Contam. Toxicol. 6:421-433.

Ghassemi, M., L. Fargo, P. Painter, S. Quinlivan, R. Scofield, and A. Takata. 1981. Environmental Fates and Impacts of Major Forest Use Pesticides. Prepared for U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. TRW Environmental Division, Redondo Beach, CA.

Hall, R.J. 1980. Effects of Environmental Contaminants on Reptiles: A Review. U.S. Department of Interior, Fish and Wildlife Service Special Scientific Report, Wildlife No. 228. 12 pp.

Han, J.C.Y. 1979. Stability of (14C) fosamine ammonium in water and soils. Journal of Agricultural and Food Chemistry 27(3):564-571.

Harvey, J., J. Dulka, and J. Anderson. 1985. Properties of sulfometuron methyl affecting its environmental fate: Aqueous hydrolysis and photolysis, mobility and absorption on soils, and bioaccumulation potential. Journal of Agricultural and Food Chemistry 33:590-596.

Hudson, R.H., R.K. Tucker, and M.A. Haegle. 1984. Handbook of Toxicity of Pesticides to Wildlife. 2nd ed. U.S. Department of Interior, Fish and Wildlife Service, Washington, DC.

Johnson, C.R. 1976. Herbicide toxicities in some Australian anurans and the effect of subacute dosages on temperature tolerance. Zoological Journal of the Linnean Society 59:79-83.

Mabey, W.R., J. Smith, R.T. Podoll, H.L. Johnson, T. Mill, T.W. Chou, J. Gates, I. Waight Partridge, H. Jaber, and D. Vanderberg. 1982. Aquatic fate process data for organic priority pollutants: Final report. Prepared by SRI International for the U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, DC. EPA Contract No. 8-01-3867/68-03-2981.

Mayer, F.L., Jr. and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication 160. U.S. Department of Interior, Fish and Wildlife Service, Washington, DC.

Rhodes, R.C. 1980. Studies with 14C-labeled hexazinone in water and bluegill sunfish. Journal of Agricultural and Food Chemistry 28:306-310.

Scott, V.E., K.E. Evans, D.R. Patton, C.P. Stone, A. Singer. 1977. Cavity-Nesting Birds of North American Forests. USDA Forest Service Agric. Handbook 511, 112 pp.

Seehorn, M.E. 1982. Reptiles and Amphibians of Southeastern National Forests. U.S. Department of Agriculture. Forest Service, Atlanta, Georgia. 85 pp.

U.S. Department of Agriculture, Forest Service. 1984. Pesticide Background Statements, Volume I: Herbicides. Agriculture Handbook No. 633. U.S. Government Printing Office, Washington, DC.

U.S. Department of Agriculture, Forest Service. 1986. Pesticide Background Statements, Volume I: Herbicides (Supplement). Agriculture Handbook No. 633. U.S. Government Printing Office, Washington, DC.

U.S. Environmental Protection Agency, Office of Pesticide Programs. 1986. Hazard Evaluation Division Standard Evaluation Procedure, Ecological Risk Assessment. EPA-540/9-85-001. Washington, DC.

SUPPLEMENT No. 1

MINERAL OILS: A REVIEW OF THEIR TOXIC PROPERTIES AND ENVIRONMENTAL FATE CHARACTERISTICS

March 30, 1989

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INTRODUCTION

This report describes the toxic and environmental fate properties of mineral oils. The next section describes the chemical and physical properties of mineral oils. The third section discusses the toxic properties of mineral oils in humans and laboratory animals. The final section discusses the environmental fate properties of mineral oils.

Section 1. CHEMICAL AND PHYSICAL PROPERTIES OF MINERAL OILS

Mineral oils are mixtures of liquid hydrocarbons produced from crude petroleum oils through a series of highly complex processes. In the past, the term "mineral oils" has been used to describe oils derived from coal, shale, petroleum crude oil, and animal and vegetable sources and products ranging from gasoline to asphalt have been referred to as "mineral oils". More recently the term "mineral oils" has been applied to lubricating oils.

The principal uses of mineral oils are as follows: pharmaceutical preparations, cosmetics, food applications, animal feed products, cosmetics, textile-machine lubricants, solvents for pesticides, turbine oils, hydraulic oils, transformer oils, automotive motor oils, and metalworking oils.

The International Agency for Research on Carcinogens (IARC, 1984) stated that "In view of problems of nomenclature and of the wide differences in the production, uses and chemical, physical and toxicological characteristics of petroleum refinery materials, a system for defining petroleum crude oil refinery process streams was adopted in 1978 under the U.S. Toxic Substance Control Act (TSCA) and in 1981 under the Commission of the European Communities' Sixth Amendment to the Dangerous Substances Directive - European Inventory of Existing Commercial Chemical Substances (EINECS). Each stream is defined on the basis of petroleum crude oil type, viscosity and process, and is identified by a Chemical Abstract Services (CAS) Registry Number, which identifies its last refining process."

The important refinery streams (most of which are used as lubricant base oils) that are included on the TSCA inventories of chemical substances in the United States and Europe and the products derived from them have been divided into eight classes based on increasing severity of processing or refinement by IARC:

Class 1. Vacuum distillates: These may have undergone subsequent finishing steps, such as caustic neutralization, dewaxing, clay treatment and/or mild hydrotreatment. They have not been acid treated or solvent extracted.

Class 2. Acid-treated oils: These may have undergone subsequent finishing steps, such as caustic neutralization, dewaxing, clay treating and/or mild hydrotreatment. They have not been solvent extracted.

Class 3. Solvent-refined-oils (raffinates): These may have undergone subsequent finishing steps, such as dewaxing, clay treating and/or mild hydrotreatment.

Class 4. Hydrotreated oils

Class 5. White oils and petrolatums suitable for food and/or medicinal use

Class 6. Aromatic oils
  6.1 Solvent extracts
  6.2 Catalytically cracked oils

Class 7. Miscellaneous materials
  7.1 Formulated products
  7.2 Used oils

Class 8. Petroleum-derived materials not otherwise classified (not sufficiently described to permit assignment to other classes)

Mineral oils are colorless, oily liquids, that are practically tasteless, and odorless; insoluble in water and alcohol but soluble in benzene, chloroform, and ether (Sax 1984).

Synonyms for mineral oils include petroleum distillate, petroleum hydrocarbon, paraffin oil, liquid petrolatum, white mineral oil, and technical white oil.

Mineral oils undergo a wide variety of chemical reactions because they contain high molecular-weight paraffinic, cycloparaffinic, and aromatic hydrocarbons. The reactions include oxidation, hydrogenation, halogenation, and sulfonation.

The physical properties of mineral oils are summarized in table 1-1.

Table 1-1--Physical Properties of Mineral Oils (white oils)

Physical State	:	oily liquid
Color	:	colorless
Odor	:	none
Viscosity (mm^2/sec) at 40° C	:	14.2-68.7
Flash Point (°C)	:	195-230
Density (kg/l) at 15 °C	:	0.843-0.890
Solubility	:	Insoluble in water and alcohol, soluble in benzene, chloroform, and ether

Sources: Sax 1984; IARC 1984

Medicinal white oils (class 5) are highly refined colorless oils free from unsaturated compounds and other constituents that influence color, odor, taste, and acceptability as a pharmaceutical and food-grade material (World Health Organization 1982). They contain hydrocarbons predominantly with carbon numbers in the range of 15 to 50 (US EPA 1978). Technical-grade white oils are less refined than medicinal oils

Section 2. TOXICITY OF MINERAL OILS

Toxicity Summary of Mineral Oils

Based on acute oral LD_{50s} of greater than 25 ml/kg in rats and acute dermal LD_{50}s of greater than 5 g/kg in rabbits, mineral oils can be classified as very slightly toxic (Maxwell 1982, as cited in Walstad and Dost 1984). Mineral oils are slight skin irritants but are not skin sensitizers. Mineral oils are also not eye irritants. Available data indicate that mineral oils are not mutagenic. There is no evidence for carcinogenicity in experimental animals of solvent-refined oils, white oils and petrolatum. However, there is some evidence for carcinogenicity of less refined oils in animals.

An association between exposure to mineral oil and the development of occupational skin cancer in man has been established. However, it has been demonstrated that the refining process influences the carcinogenic potential of mineral oil. Most lubricating mineral oil in use today is not carcinogenic because of newer refining techniques (such as hydrotreatment or solvent extraction) as assayed by mouse skin painting. In contrast, oils refined by the older acid/earth process are carcinogenic. Therefore, these facts must be borne in mind when considering epidemiology studies of workers exposed to lubricating oils prior to or for some years after World War II.

The majority of both human and laboratory animal studies discussed below were taken from the Monographs of International Agency for Research on Cancer (IARC, 1984).

Mineral Oil Toxicity in Humans

General Toxic Effects

The toxic effects of petroleum-derived oils in humans were reviewed by Jampolis et al., 1953, Key et al., 1966, Kipling 1967, and Hodgson 1970 as cited in IARC (1984).

The major sites of the toxicity of petroleum-derived oils are the skin and the lungs. Toxic effects observed on the skin are eczematous dermatitis, contact dermatitis, folliculitis, oil acne, lipid granuloma and melanosis (Hodgson 1970 as cited in IARC). Habitual use of large amounts of mineral oil (paraffin oil or liquid petrolatum of class 5) suitable for food and medicinal use by nasal, oral, or pharyngeal administration for prolonged periods of time result in lipid granuloma (localized lipid pneumonia) of the lung (Jampolis et al., 1953, as cited in IARC 1984).

The occupational exposure limit for mineral-oil mist in the U.S. is 5 mg/m^3 on an eight-hour time-weighted average (TWA). The American Conference of Governmental Industrial Hygienists (1982) recommends a short-term exposure limit (STEL) of 10 mg/m^3.

The U.S. Environmental Protection Agency (EPA) has established tolerances for total residues of mineral oil (class 5) of 0.2 g/kg in or on shelled corn and grain sorghum from post-harvest applications. Mineral oil-class 5 is exempted from the requirement of a tolerance when used as an ingredient in formulations of pesticidal products for growing crops or for raw agricultural commodities after harvest (US EPA 1982a).

Mineral oil-class 5 is approved for use by the FDA under 21 CFR, Part 172.878 as a direct multipurpose food additive and Part 573.680 in animal feed, subject to the provisions of these subparts.

Metabolism

In general, the absorption of mineral oil-class 5 from the gastrointestinal tract is limited (Anon 1967; Goodman and Gilman 1975 as cited in IARC 1984).

Mutagenicity

The urine of 16 men exposed occupationally to mineral oil-class 8 was tested for mutagenicity in Salmonella typhimurium strains TA98 and TA100 in the presence of rat-liver metabolic system. These workers exhibited mutagenic activity in their urine.

Carcinogenicity

Human exposure to the mineral oils (lubricant base oils and derived products excluding class 5) varies widely. Among occupations, including mulespinning, metal machine and jute processing, exposure to these materials has been associated with the occurrence of squamous-cell cancers of the skin, and especially of the scrotum.

The epidemiological studies of metal industry workers reported excessive gastrointestinal malignancies in each of the three cohort studies. Also excesses of bladder cancer have been reported in case-control studies in several countries among machinists and engineers who were exposed to cutting oils (containing aromatic amines).

Mineral Oil Toxicity in Laboratory Animals

General Toxic Effects

Toxic effects of mineral oils in animals are reviewed by Bingham et al., 1980, Chircova 1982, and World Health Organization 1982 as cited in IARC (1984).

No alteration in respiratory function was observed in guinea pigs exposed to mineral oil-class 5 at concentrations of 10 or 40 mg/m^3 for one hour (Costa and Amdur 1979, as cited in IARC 1984). Aspiration of 0.2 ml mineral oil-class 5 by rats caused no mortality after 24 hours. This oil did not produce the severe acute pulmonary edema or hemorrhage characteristic of kerosene (Gerarde 1963 as cited in IARC 1984). A survival rate of 77 percent was observed in 13 mice exposed to an aerosol of mineral oil (US Pharmacopeia grade liquid petrolatum-class 5) at a concentration of 4,500 mg/m^3 intermittantly for a total of 80-84 hours over a 4-week period (Shoshkes et al., 1950 as cited in IARC 1984). This oil produced localized foreign-body reactions in the lung as well as patches of lipid pneumonia.

No toxic effects were observed in rats receiving mineral oil-class 5 at an oral (gavage) dose of 2 ml/kg three times weekly for three months (Kimborough et al. 1980, as cited in IARC 1984). Beck et al. 1983 (as cited in Kane et al., 1984) showed that solvent-refined naphthenic oil-class 3 has a low order of acute toxicity. This oil had an oral LD_{50} value of greater than 25 ml/kg and dermal LD_{50} value of greater than 5 g/kg, was slightly irritating to skin based on the primary dermal irritation test but was not a skin sensitizer based on the guinea pig skin sensitization test, and not irritating to eyes based on the primary eye irritation test.

Metabolism

Ebert et al. 1966 (as cited in IARC 1984) orally administered radio-labelled mineral oil-class 5 to rats at 0.66 ml/kg. Five hours after oral administration, 1.5 percent of the administered dose was found in the carcasses as non-mineral oil substances. However, liver, fat, kidney, brain, and spleen contained mineral oil (the amount of oil after administration was not specified). Within 2 days after administration, the amount of mineral oil found in the animals was 0.3 percent of administered dose.

Mutagenicity

No mutagenic activity was found when mineral oil-class 5 was tested for mutagenicity in S. typhimurium strain TA98 in the presence of rat liver S9 metabolic system (Hermann et al. 1980 a, b).

Carcinogenicity

In a 500-day feeding study, rats received 2 percent liquid paraffin-class 5 in the diet. The total dose received was 136 ml per animal. No significant tumor induction was observed (Schmähl and Reiter 1953 as cited in IARC 1984).

Groups of 50 male and 50 female rats were fed three samples of petrolatum (class 5) (snow-white USP XVI grade, white USP XVI grade, and yellow National Formulary XI grade) at a concentration of 5 percent in their diet for 2 years. No significant tumor induction was observed in these tests at the dose studied (Oser et al. 1965, as cited in IARC 1984).

Mouse skin tumorigenicity studies of mineral oil of different classes were reported by Kane et al., 1984. Male C3H or C3H/HeJ mice received skin applications of 25-100 mg or 0.2 ml of test materials 2 or 3 times each week for 80 weeks until a tumor was grossly diagnosed or for lifetime of the animals until an advanced tumor was grossly diagnosed. This study showed that unprocessed refinery distillates (class 1) caused tumors and solvent refining removed the tumorigenic components. The results obtained by Halder et al. 1984 (as cited in IARC 1984) confirmed the work of Kane et al. (1984).

Groups of 27 or 50 mice were given skin applications of 12 mineral oils once or twice weekly or once every two weeks for 78 weeks (Doak et al. 1983). The amount of test materials applied was 0.25 ml for the first 22 weeks followed by 0.2 ml. Malignant skin tumors were found in 4/27 animals painted acid-treated naphthenic distillate (class 2). Among the 11 oils processed by other refining routes, 2 oils were less carcinogenic and 9 oils including white oil (class 5) which had been solvent-refined were non-carcinogenic to skin.

A group of 130 mice were exposed daily to an aerosol (mean particle diameter, 1.3 μm) generated from a light white naphthenic oil (class 5) at a concentration of 100 mg/m^3 for 7-13 months. Animals were sacrificed at monthly intervals. No significant tumor induction was observed (Wagner et al. 1964, as cited in IARC 1984). These authors also tested this material in rabbits, rats, hamsters, and dogs by exposing them to concentrations of 5 and 100 mg/m^3 for periods of 6-26 months. No tumors were reported in the treated animals.

A summary of mouse skin tumorigenicity presented in IARC 1984 is shown in table 2-1. There is sufficient evidence for the carcinogenicity in experimental animals of vacuum distillates (class 1), acid-treated oils (class 2), mildly solvent-refined oils (class 3), mildly hydrotreated oils (class 4), and aromatic oils (class 6). There is no evidence for carcinogenicity in experimental animals of severely solvent-refined oils (class 3) and white oils and petrolatums (class 5). However, the data are inadequate to evaluate severely hydrotreated oils (class 4), formulated products (class 7.1), and used oils (class 7.2).

Table 2-1. Summary of Tumorigenicity in Experimental Animals

IARC Classification		Tumorigenicity
Class 1.	Vacuum distillates	Sufficient evidence
Class 2.	Acid-treated oils	Sufficient evidence
Class 3.	Solvent-refined oils	
	Mildly treated	Sufficient evidence
	Severely treated	No evidence
Class 4.	Hydrotreated Oils	
	Mildly treated	Sufficient evidence
	Severely treated	Inadequate evidence
Class 5.	White oils and petrolatums	No evidence
Class 6.	Aromatic oils	Sufficient evidence
Class 7.	Miscellaneous materials	
	7.1 Formulated products	Inadequate evidence
	7.2 Used oils	Inadequate evidence

Section 3. ENVIRONMENTAL FATE PROPERTIES OF MINERAL OILS

Mineral oils are mixtures of a variety of long-chain hydrocarbons (chemicals containing primarily carbon and hydrogen). While the environmental fate properties of a large number of individual chemicals have been studied, the fate properties of the longer-chain hydrocarbons have not been well studied. Environmental fate information does not appear to be available on individual large hydrocarbons. Defining the fate properties of a poorly defined mixture of these larger hydrocarbons would be difficult even if the requisite information existed for the individual hydrocarbon constituents.

Literature information on the environmental fate properties of octane and of diesel oil and kerosene is available to a limited degree. Octane is an 8-carbon simple chain hydrocarbon. Diesel oil and kerosene, like mineral oils, are mixtures of hydrocarbon compounds. Octane and the hydrocarbons found in diesel oils and kerosene are shorter in length and lighter than those in mineral oils. Based on the differences between the composition octane and of of diesel oil and kerosene and the composition of mineral oils, it is possible to draw inferences about the environmental fate properties of mineral oils relative to octane, diesel oils, and kerosene.

Water Solubility

The water solubility of octane is 0.66 mg/L at 25 C. Octane should be expected to be more soluble in water than mineral oils due to its smaller molecular size and weight.

Bioaccumulation

A measure of the affinity of a chemical for water versus an organic solvent is the octanol-water partition coefficient (K_{ow}). Measurement of this coefficient is performed by adding the test chemical to a system which contains water and octanol, which are immiscible. Compounds which readily dissolve in the water phase have a very low K_{ow} value. Conversely, compounds which more readily dissolve in the octanol phase have a high K_{ow} value.
A direct relationship exists between the number of carbon atoms and the K_{ow} of a compound. The more carbons, the less soluble in water and the higher the K_{ow}. The K_{ow} of the 8-carbon octane is approximately 10,000 (Mabey et al., 1982). In mineral oils, the hydrocarbon components generally fall in the range of 15 to 50 carbon atoms. Therefore, the K_{ow} of mineral oil is expected to be greater than 10,000.

The K_{ow} value is often used as a measure of the bioaccumulative tendency of a chemical. Organisms contain a large proportion of lipids (fats and oils), and chemicals which have high K_{ow} values are more likely to accumulate in these lipid materials. The ability of a chemical to bioaccumulate is sometimes measured experimentally, but is more often calculated by a formula relating K_{ow} to the bioconcentration factor (BCF). A common formula for performing this calculation is given by Veith et al. (1979 in Lyman et al., 1982):

$$\log BCF = 0.76 \log K_{ow} - 0.23$$

Using the Veith equation, the calculated BCF of octane is 646. Mineral oils, because of their expected higher K_{ow}, would be expected to have higher BCF values, that is, they would tend to more readily concentrate in animal lipid tissues.

Fate in Soil

Octane adsorbs readily to soils, with a distribution coefficient (K_d) of 110 L/g (calculated by Lyman et al., 1982). Because of the larger carbon structures and lower water solubilities, the components of mineral oil will be more likely to adsorb to soils than octane.

Vapor Pressure

Diesel oil exhibits a vapor pressure of 2.07 mm Hg at 40 C. Owing to the lighter, more volatile components of diesel oil, mineral oil should have a lower vapor pressure and therefore be less volatile.

Biodegradation

The biodegradation constant of octane is approximately 0.11 day_{-1} (Ladd, 1956). Because of the lower relative water solubility of mineral oils (chemicals must be dissolved in water to be consumed by microorganisms), the greater relative soil adsorption of mineral oils (chemicals adsorbed to soil are unavailable for biodegradation and are less likely to partition to water), and the larger molecular size and weight, mineral oil components would be expected to have a biodegradation rate much lower than octane.

LITERATURE CITED

American Conference of Governmental Industrial Hygienists. 1982. *Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1982*, Cincinnati, OH, p. 26; as cited in IARC 1984.

Anon. 1967. Mineral oil in human tissues. *Nutr. Rev*., 25, 46-49; as cited in IARC 1984.

Beck, L.S., Hepler, D.I., Hansen, K.L. 1983. The acute toxicology of selected petroleum products. In MacFarland H.N., Holdsworth, C.E., MacGregor, J.A., Kane, M.L. eds, *Applied Toxicology of Petroleum Hydrocarbons*. Princeton, New Jersey. Princeton Scientific Press, as cited in Kane et al., 1984.

Bingham, E., Trosset, R.P. and Warshawsky, D. 1980. Carcinogenic potential of petroleum hydrocarbons. A critical review of the literature. *J. environ. Pathol. Toxicol*. 3, 483-563; as cited in IARC 1984.

Chircova, E.M. 1982. *Mineral oils*. In: Izmerov, N.F., ed., *Mineral Oils*, Moscow, Centre of International Projects, State Committee on the Science and Technique, pp. 2-14; as cited in IARC 1984.

Costa, D.L. and Amdur, M.O. 1979. Respiratory response of guinea pigs to oil ists. *Am. Ind. Hyg. Assoc. J*., 40, 673-679; as cited in IARC 1984.

Doak, S.M.A., Brown, V.K.H., Hunt, P.F., Smith, J.D. and Roe, F.J.C. 1983. The carcinogenic potential of twelve refined mineral oils following long-term topical application. *Br. J. Cancer*, 48, 429-436; as cited in IARC 1984.

Ebert, A.G., Schleifer, C.R. and Hess, S.M. 1966. Absorption, disposition, and excretion of ^{3}H-Mineral oil in rats. *J. Pharm. Sci*., 55, 923-929; as cited in IARC 1984.

Gerarde, H.W. 1963. Toxicological studies on hydrocarbons. IX. The aspiration hazard and toxicity of hydrocarbons and hydrocarbon mixtures. *Arch. Environ. Health*, 6, 329-341; as cited in IARC 1984.

Goodman, L.S. and Gilman, A. 1975. *The Pharmacological Basis of Therapeutics*, 5th ed., New York. Macmillan, p. 978; as cited in IARC 1984.

Halder, C.A., Warne, T.M., Little, R.Q. and Garvin, P.J. 1984. Carcinogenicity of petroleum lubricating oil distillates. 1. Effects of solvent-refining, hydroprocessing, and blending. *Am. J. Ind. Med*. (in press); as cited in IARC 1984.

Hermann, M., Chaudé, O., Weill, N., Bedouelle, H. and Hofnung, M. 1980a. Adaptation of the *Salmonella*/mammalian microsome test to the determination of the mutagenic properties of mineral oils. *Mutat. Res*., 77, 327-339; as cited in IARC 1984.

Hermann, M., Durand, J.P., Charpentier, J.M., Chaudé, O., Hofnung, M., Pétroff, N., Vandecasteele, J.P. and Weill, N. 1980b. *Correlations of mutagenic activity with polynuclear aromatic hydrocarbon content of various mineral oils*. In: Bjørseth, A. and Dennis, A.J., eds, *Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects. 4th Int. Symp*., Columbus, OH. Battelle Press, pp. 899-916.

Hodgson, G. 1970. Cutaneous hazards of lubricants. *Ind. Med*., 39, 68-73; as cited in IARC 1984.

International Agency for Research on Cancer (IARC) 1984. *Polynuclear Aromatic Hydrocarbons, Part 2, Carbon Blacks, Mineral Oils (Lubricant Base Oils and Derived Products) and some Nitroarenes; IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*. 33 Lyon: International Agency for Research on Cancer.

Jampolis, R.W., McDonald, J.R. and Clagett, O.T. 1953. Mineral oil granuloma of the lungs: An evaluation of methods for identification of mineral oil in tissue. *Int. Abstr. Surg*., 97, 105-119; as cited in IARC 1984.

Kane, M.L., Ladov, E.N., Holdsworth, C.E. and Weaver, N.K. 1984. Toxicological characteristics of refinery streams used to manufacture lubricating oils. *Am. J. Ind. Med*., 5, 183-200.

Key, M.M., Ritter, E.J. and Arndt, K.A. 1966. Cutting and grinding fluids and their effects on the skin. *Am. Ind. Hyg. Assoc. J*., 27, 423-427; as cited in IARC 1984.

Kimbrough, R.D., Korver, M.P., Burse, V.W. and Groce, D.F. 1980. The effect of different diets or mineral oil on liver pathology and polybrominated biphenyl concentration in tissues. *Toxicol. Appl. Pharmacol*., 52, 442-453; as cited in IARC 1984.

Kipling, M.D. 1967. *Oil and the skin.* In: *Annual Report of H.M. Chief Inspector of Factories*, London. Her Majesty's Stationery Office, pp. 105-119; as cited in IARC 1984.

Ladd, J.N. 1956. The oxidation of hydrocarbons by soil bacteria: I. Morphological and biochemical properties of a soil Diptheriod utilizing hydrocarbons. *Australian Journal of Biological Sciences* 9:92.

Laires, A., Borba, H., Rueff, J., Gomes, M.I. and Halpern, M. 1982. Urinary mutagenicity in occupational exposure to mineral oils and iron oxide particles. *Carcinogenesis*, 3, 1077-1079; as cited in IARC 1984.

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill, Inc., New York.

Mabey, W.R., J.H. Smith, R.T. Podoll, H.L. Johnson, T. Mill, T.W. Chou, J. Gates, I. Waight Partridge, H. Jaber, and D. Vanderberg. 1982. Aquatic fate process data for organic priority pollutants: final report. Prepared by SRI International for the U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, DC. EPA Contract No. 68-01-867/68-03-2981.

Maxwell, R.C. 1982. Meaning of label signal words. Pesticide Report 43:4-5. Washington State University, Cooperative Extension Service, Pullman, WA; as cited in Walstad and Dost 1984.

Merck Index. 1983. 10th ed., p 1033.

Oser, B.L., Oser, M., Carson, S. and Sternberg, S.S. 1965. Toxicologic studies of petrolatum in mice and rats. *Toxicol. Appl. Pharmacol*., 7, 382-401.

Sax, N.I. 1984. *Dangerous Properties of Industrial Materials*. 6th ed. Van Nostrand Reinhold Co., N.Y.

Schmahl, D. and Reiter, A. 1953. Production of tumours with liquid paraffin, yellow petrolatum and lanolin (Ger.). *Arzneimittel-Forsch*., 3, 403-406; as cited in IARC 1984.

Shoshkes, M., Banfield, W.G., Jr., Rosenbaum, S.J. and Fisk, A.J. 1950. Distribution effect and fate of oil aerosol particles retained in the lungs of mice. A.M.A. Arch. Ind. Hyg. Occup. Med., 1, 20-35; as cited in IARC 1984.

U.S. Environmental Protection Agency. 1978. Toxic Substances Control Act (TSCA) PL94-469, Candidate List of Chemical Substances, Addendum I, Generic Terms Covering Petroleum Refinery Process Streams, Office of Toxic Substances, Washington D.C.

U.S. Environmental Protection Agency. 1982. Tolerances and exemptions from tolerances for pesticide chemicals in or on raw agricultural commodities. U.S. Code Fed. Regul., Title 40, Parts 180.149, 180.1001(c), 180.1001(e), pp. 158-165, 196, 201, 265, 275.

Veith, G.D., K.J. Macek, S.R. Petrocelli and J. Carroll. 1980. An Evaluation of Using Partition Coefficients and Water Solubiity to Esitmate Bioconcentration Factors for Organic Chemicals in Fish. Journal of the Fisheries Research Board of Canada (prepublication copy).

Wagner, W.D., Wright, P.G. and Stokinger, H.E. 1964. Inhalation toxicology of oil mists. I. Chronic effects of white mineral oil. Am. Ind. Hyg. Assoc. J., 25, 158-168; as cited in IARC 1984.

Waldstad, J.D. and Dost, F.N. 1984. The Health Risks of Herbicides in Forestry: A Review of the Scientific Record. Forest Research Station, Oregon State University. Special Publication 10.

World Health Organization. 1982. Selected Petroleum Products: Environmental Health Criteria 20, Geneva, pp. 62-79.; as cited in IARC 1984.

Effects of Prescribed Fire on Soil and Water in Southern National Forests

APPENDIX B

APPENDIX B

EFFECTS OF PRESCRIBED FIRE ON SOIL AND WATER IN SOUTHERN NATIONAL FORESTS

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EFFECTS OF PRESCRIBED FIRE ON SOIL AND WATER IN SOUTHERN NATIONAL FORESTS

Abstract

Effects of prescribed fire on soil and water vary with type of burn. Slash burns can reduce soil quality if the burn is severe, consuming all litter and duff and altering mineral soil on much of the area. Major effects are loss of soil biota, structure, organic matter, and nitrogen. Severe burns also yield high sediment loads in rugged terrain. On poor soils, moderate burns may prevent buildup of organic matter and nitrogen vital to site recovery. Underburns more often than every 3 years can reduce soil quality via loss of soil organisms and organic matter. On poor soils, burns more often than every 5 years may prevent buildup of organic matter vital to site recovery. Unless alternated with winter burns, summer burns can cause excessive nitrogen loss. Underburns can enhance soil quality by retarding soil weathering. Grassland burns cause few impacts if done less often than every 2-3 years.

INTRODUCTION

Prescribed fire is a much-used management tool in southern forests. The main types are: (1) slash burns in harvested stands; (2) underburns beneath stands; and (3) grass burns. Slash burns consume litter, brush, limbs, and foliage and can be severe if done in hot, dry weather (Pritchett 1976). Underburns and grass burns generally consume litter and low-growing vegetation.

Effects of prescribed fire on soil and water depend on fire severity and frequency and on soil and site properties. Fire severity is determined by fire duration and intensity, which depend on fuel characteristics, topography, and weather. Important soil and site properties are soil moisture and nutrient content, soil erodibility, and topography.

Prescribed fire can impact soil and water by: (1) soil heating which can kill soil biota, alter soil physics, consume organic matter, and release nutrients; and (2) soil exposure which can increase runoff and erosion. These effects can vary greatly with fuel, site, and burning conditions, so they are expressed as degrees of risk rather than absolute predictions.

FIRE SEVERITY

Fire severity is defined by condition of the ground surface after a burn (Wells and others 1979). Fire severity for specific burned spots is classed as:

LIGHT--litter and duff scorched but not altered through entire depth
MODERATE--litter and duff charred; mineral soil not visibly altered
SEVERE--litter and duff consumed; color-structure of soil visibly altered

Fire severity for a total burned area is classed as:

LIGHT--less than 2 percent severe, less than 15 percent moderate
MODERATE--less than 10 percent severe, more than 15 percent moderate
SEVERE--more than 10 percent severe or more than 80 percent moderate-severe

Fire severity reflects degree of soil heating and exposure. Slash burns occur every 40-90 years and can consume all litter and duff and alter mineral soil on much of the area. Risks of adverse soil and water effects depend on severity of burn. Underburns and grass burns occur every 1-7 years and usually leave a scorched or charred litter surface. Risks depend more on frequency of burn.

SOIL HEATING BY PRESCRIBED FIRE

Soil heating is less obvious than erosion but is more important for long-term productivity. Adverse effects increase with fire severity and frequency. Excess heating can kill soil biota, alter soil physics, consume organic matter, and release site nutrients.

Fire intensity reflects a fire's heat output. It increases with higher fuel loads and lower fuel moisture, higher wind and temperature and lower humidity, and steeper slopes. But fire intensity is not the key factor affecting a fire's impacts on a given soil. Duration of heating determines depth of heating. An intense fire consuming small fuels may briefly produce temperatures of 500-700 C but not penetrate the insulating duff. In general, large fuels must burn to produce heating with depth and cause severe impacts (Martin 1981).

Litter and duff insulate soil against heating (Wells and others 1979). An underburn in California produced temperatures of 260 C in litter but only 93 C at the soil surface (Agee 1973). In a Canadian underburn, temperature decreases per 0.1 inch of soil depth were 25 C in bare soil and 70 C under duff. Soil heating is minimal when 0.5 inch of duff remains unburned (Van Wagner 1970).

Soil heating is minimal when duff and soil are moist (Barber and Van Lear 1984; DeBano and others 1979; Frandsen and Ryan 1986). The temperature of any soil layer can't exceed 100 C until all moisture is evaporated (DeBano and others 1976). Large fuels are generally moist and unavailable when duff is moist, so burning then limits both surface heating and soil penetration (Martin 1981).

Soil Heating by Slash Burns

Data on soil heating by slash burns in the South are lacking. In the Pacific Northwest, severe slash burns have attained surface temperatures of 538-1260 C (Barnett 1984). In one burn with soil moisture of 16-20 percent, maximum temperatures were 538 C at 0.2 inch and 340 C at 1 inch (Neal and others 1965). Slash burns with soil moisture of 60-95 percent produced temperatures exceeding 149 C at 1.4 inches on 77 percent of plots (Barnett 1984). Isaac and Hopkins (1937) measured 320 C at 1 inch under a burned slash pile.

Australian slash burns (Humphreys and Lambert 1965) created surface temperatures of 360 C in light slash, 400 C in medium slash, 480 C in heavy slash, and up to 900 C in slash piles. Slash fires and wildfires have produced temperatures at 1 inch of 90-114 C (Beadle 1940; Humphreys and Lambert 1965). Burning windrows and slash piles over dry soil produced temperatures of 180-580 C at 1 inch and 120-338 C at 2 inches (Beadle 1940; Humphreys and Lambert 1965; Powers 1965; Cromer and Vines 1966).

In Canada, a burning lumber pile produced maximum surface temperatures of 680 C on dry sand, 400 C on dry sand under moss, and 90 C on wet sand under wet moss. Maximum temperatures at 0.8 inch were 420 C in dry sand, 260 C in dry sand under moss, and 80 C in wet sand under wet moss (Frandsen and Ryan 1986).

Chaparral heating curves compute maximum surface temperatures of 700 C in severe burns, 425 C in moderate burns, and 250 C in light burns, and soil temperatures in moderate burns of 199 C at 1 inch and 80 C at 2 inches (DeBano and others 1979). A fire producing 716 C at the surface heated soil to 270 C at 0.2 inch, 166 C at 1 inch, and 66 C at 2 inches (DeBano and Rice 1971; Scholl 1975). Prescribed fires have created temperatures of 200 C and 400 C at 0.2 inch under standing and crushed brush (Scott and Burgy 1956), 260 C at 0.8 inch under dry slash (Dunn and DeBano 1977), and 149 C at 1.5 inches (Sampson 1944).

Soil heating by slash burns varies with fuel properties and soil moisture. No data exist for the South, but the above data do give some idea of temperatures likely for certain fuels, moisture levels, and burning regimes. Southern fuel loads are usually less than those in the Pacific Northwest. Heating data from chaparral burns, with fuel loads often less than 20 tons per acre and concentrated in the brush canopy, were used to approximate southern burning conditions. Typical temperatures (C) by fire severity class are estimated to be:

	Surface	0-1 in	0-2 in
Light	250	100	40
Moderate	400	200	90
Severe	700	330	150

Soil Heating by Underburns

In South Carolina flatwoods, surface temperatures reached 70 C in a headfire and 150 C in a backfire (Lindenmuth and Byram 1948). In south Georgia, they were 90-150 C with wet duff, 135-190 C with moist duff, and 175-230 with dry duff (Harshbarger and others 1975).

Soil temperatures under longleaf pine reached 135 C at 0.2 inch and 66 C at 1 inch (Heyward 1938). An Australian underburn produced 452 C at the surface, 57 C at 0.8 inch, and 37 C at 2 inches (Raison and others 1986). Temperatures rarely exceed 100 C at 0.1 inch (Heyward 1936); at 1-2 inches, they seldom exceed 52 C for more than 15 minutes and never exceed 120 C except for 2-3 minute intervals (Pritchett 1976). Underburns, which consume only small fuels and usually retain some duff, are light to moderate.

Soil Heating by Grass Burns

Grass fires have produced surface temperatures of 40-150 C in Texas (Fonteyn and others 1984), 177 C in California (Bentley and Fenner 1958) and 245 C in Australia (Tothill and Shaw 1968). A winter burn in Texas prairie achieved 400-450 C with consumption of 95 percent of mulch loads (Mutz and others 1985).

Bentley and Fenner (1958) measured the following maximum surface and soil temperatures (C) for 3 post-fire ground conditions in California savannah:

	Surface	0.3 in	1.0 in
Charred litter	177	121	71
Bare soil	399	288	177
White ash	510	399	288

Bentley and Penner measured 93 C at 0.5 inch for the charred litter condition, while Tothill and Shaw (1968) measured 68 C at 0.5 inch for a grass fire in Australia with a maximum surface temperature of 245 C. Grass burns are usually light to moderate.

EFFECTS ON SOIL BIOTA

Soil flora and fauna enhance soil physics and biochemical processes. Duration of heating, maximum temperatures, and soil moisture most affect biotic response (Wells and others 1979). Recovery is usually rapid, but species composition can change if litter-duff or soil properties are changed (Henley and Clarke 1976).

Soil Flora

At 120 C in dry soil and 60 C in wet soil, normal saprophytic fungi are replaced by "heat shock" fungi, which are then killed at 155 C in dry soil and 100 C in wet soil. Most bacteria are killed at 210 C in dry soil and 110 C in wet soil. Nitrifying bacteria, important because they mineralize nitrogen from organic to inorganic (ammonium and nitrate) forms available to plants (Wells and others 1979), are particularly sensitive to soil heating. Lethal temperatures are 140 C in dry soil and 75 C in wet soil for Nitrosomonas and 100 C in dry soil and 50 C in wet soil for Nitrobacter (Dunn and DeBano 1977). Nitrogen-fixing and nitrifying bacteria can recover quickly and are favored by the increased pH, temperature, and nutrient availability prevalent after a fire (Feller 1982).

Light to moderate burns little affect soil microflora because minor changes in soil properties occur (Wells and others 1979). Severe burns can temporarily sterilize the soil. Winter underburns in South Carolina flatwoods reduced litter and duff by 68 percent and their microflora by 82 percent after 1 year, but microflora recovered quickly and exceeded numbers on unburned plots when checked after 8 years. Soil microflora were not affected (Jorgensen and Hodges 1971). Ahlgren and Ahlgren (1965) found bacterial numbers reduced significantly by heating to 200 C for 25 minutes. Slash burning reduced mycorrhizae occurrence on Douglas-fir seedlings (Tarrant 1956b), but 1-7 year underburns in South Carolina flatwoods did not (Kinnes 1982). Laboratory heating of topsoil eliminated 99 percent of fungi, nitrite oxidizers, and bacteria at 80-110 C (Dunn and others 1985). In dry soil (less than 5 percent moisture), microbes increased from heating to 60 C and then declined.

Soil Fauna

Activity of soil fauna is reduced by frequent underburns and recovers within 3 years (Heyward 1937). A winter underburn in South Carolina flatwoods (Metz and Farrier 1971) partly consumed litter and duff and reduced their mesofauna (mites, collembolans, insects) by 84 percent. Recovery occurred within three years. Soil mesofauna were not affected.

Underburns every 1-2 years reduce total numbers of soil fauna by 60-90 percent (Pearse 1943; Heyward and Tissot 1936). Earthworms, ants, and centipedes and millipedes are reduced by 50-75 percent. Annual underburns in Louisiana increased populations of harmful nematodes (Murad and others 1979).

Conclusions

Light burns do not heat soil enough to significantly affect soil biota. Litter biota are reduced but quickly recover. Moderate burns probably kill all litter biota and some soil biota; litter accumulation should complete recovery within three years. Severe burns sterilize a site; total recovery may require years of litter buildup (Jorgensen and Wells 1986). Underburns and grass burns should not reduce biotic activity unless done more often than every 3 years.

EFFECTS ON SOIL PHYSICS

Fire can affect soil structure, infiltration and soil moisture, and water repellency. Effects depend on fire severity and frequency.

Soil Structure

Unless a fire removes litter and duff and significant soil organic matter, soil structure should not be changed (Wells and others 1979). Severe burns reduce macropores by up to 80 percent and increase bulk density by up to 25 percent, but return of ground cover completes recovery within 4 years (Auten 1934; Bower 1966; Tarrant 1956a). Light to moderate burns do not produce these effects.

Annual underburns increase topsoil bulk density (Heyward 1936; Murad and others 1979; Wahlenberg 1935) and can reduce soil porosity (Garren 1943; Heyward 1937; Wahlenberg and others 1939). Annual summer underburns reduce topsoil macropores, but biennial underburns do not (Moehring and others 1966).

Infiltration and Soil Moisture

A burn that exposes mineral soil may reduce infiltration and moisture-holding capacity by up to 75 percent (Auten 1934; Tarrant 1956a). Soil particles are dispersed by raindrop impact and clog pores to create a surface crust that retards infiltration and aeration. Increased sunlight can add to these effects by speeding oxidation of soil organic matter (Wells and others 1979). Annual underburns reduce infiltration by 35-75 percent (Arend 1941; Kittredge 1938; Meginnis 1935; Wahlenberg 1935).

If duff is not consumed, changes are not detectable even with intense slash burns (Metz and others 1961; Moehring and others 1966; Van Lear and Danielovich 1988). Soil moisture-holding capacity does not appear to be reduced unless soil organic matter is reduced (Wells and others 1979).

Water Repellency

A water repellent soil layer can form if a fire distills organic matter downward to condense in the soil (DeBano 1966). Degree of repellency increases with hotter fire, drier soil, and coarser soil texture (Wells and others 1979). Soil temperatures exceeding 200 C can cause repellency (DeBano 1981). Repellency reduces infiltration and increases runoff and erosion (Wells and others 1979).

Conclusions

Fires exposing little mineral soil do not reduce soil porosity, infiltration, and moisture capacity. Severe fires may cause such effects and induce water repellency. Effects of a severe fire may persist for years until litter, duff, and soil organic matter recover. Underburns every 2 years or more do not impair soil physics.

EFFECTS ON ORGANIC MATTER

Organic matter is vital to the physical, chemical, and biological quality of soil (Jorgensen and Wells 1986) for 4 reasons:

1. It improves soil structure and enhances root growth, aeration, and water holding capacity.
2. It supplies most of the cation exchange (nutrient holding) capacity in forest soils.
3. It is the main source of available phosphorus and sulfur and virtually the only source of available nitrogen, and regulates nutrient release to soil through decomposition. A soil's nitrogen pool, and the effectiveness of added nitrogen, depends on its organic matter content.
4. It provides the energy source for microbes that decompose it and release nutrients vital to plant growth.

Most organic matter occurs in litter, duff, and topsoil. Litter and duff supply organic matter to soil by decomposition and build to an equilibrium about 2-3 years after grass burns, 8-10 years after underburns, and 30-35 years after slash burns (Brender and others 1976; Hough 1982; Jorgensen and Wells 1986). About 30 percent of total site organic matter often occurs in the litter-duff reserve, and 70 percent in the soil (McKee 1982).

Burning may reduce organic matter by consuming litter and duff and heating soil (Barnett 1984). A light to moderate burn may increase soil organic matter via leaching or by promoting herbaceous plants with fibrous root systems. Soil temperatures of 50-100 C cause humic acids to break down. At 100-200 C, volatile organic compounds are distilled and lignin and cellulose start breaking down, causing slight reductions in soil organic matter. Above 200 C, up to 85 percent is distilled (DeBano and others 1977; Frandsen and Ryan 1986; Hosking 1938). All humus is lost at 400 C (Wells 1981). Heating at 450 C for two hours or 500 C for 30 minutes removes 99 percent of organic matter (Hosking 1938).

Organic matter loss from soil depends on duff and soil moisture. A severe burn may aid seedling establishment and growth by removing competition and releasing nutrients, but subsequent growth may decline due to lack of organic matter and long term nutrient release (Frandsen and Ryan 1986).

Consumption of Litter and Duff

Litter and duff consumption vary with burn severity. A light slash burn in the Florida flatwoods consumed only 16,800 (21 percent) of 78,400 lb/ac (Burger and

Pritchett 1988). A winter underburn in South Carolina flatwoods (Wells 1971) consumed only 6500 (27 percent) of 24,000 lb/ac. Brender and Cooper (1968) found 4800-6300 lb/ac consumed in the Georgia Piedmont. DeBano and others (1979) estimated that a moderate burn would consume 85 percent, while a severe burn consumed all litter and duff.

Underburn frequency affects litter-duff loss. In four Coastal Plain sites, loss over time was 60-84 percent for annual burns, 61-67 percent for 2-4 year burns, and 30-33 percent for 7-year burns (McKee 1982). Annual burns reduced litter and duff more than periodic burns in Tennessee (Thor and Nichols 1974). In Arkansas, reductions were 69 percent by annual burns and 44 percent by biennial burns (Moehring and others 1966). Biennial burns in Georgia flatwoods reduced loads by nearly 80 percent (McKee and Lewis 1982). Seven annual burns in the Virginia Coastal Plain reduced them by 39 percent (Romancier 1960).

Texas grass fires with maximum surface temperatures of 400-450 C consumed 95 percent of mulch loads. Recovery took 2-3 years (Mutz and others 1985).

Changes in Soil Organic Matter

Fire affects soil organic matter through soil heating. In chaparral, a moderate burn destroyed only humic acids at 1 inch, while a severe fire heated the soil to 200 C at 1 inch and destructively distilled organic matter (DeBano and others 1979). Another fire reduced organic matter by 45 percent in litter, 19 percent in 0-0.5 inch soil, and 9 percent in 0.5-1.0 inch soil (DeBano and Conrad 1978).

Light to moderate slash burns in the Pacific Northwest did not reduce topsoil organic matter, but severe burns reduced it from 11 to 8 percent (Dyrness and Youngberg 1957). Reductions in soil organic matter by severe burns have been 75 percent in 0-0.5 inch (Austin and Baisinger 1955), 65-88 percent in 0-1 inch (Barnett 1984), 43-68 percent in 0-3 inches (Youngberg 1953), 63 percent in 1-2 inches, and 36-41 percent in 2-4 inches (Barnett 1984). Severe burns reduce organic matter in the top 2.5 inches of soil by 50+ percent (Barnett 1984).

Effects of repeated underburns on soil organic matter vary. Moehring and others (1966) found that annual and biennial underburns did not significantly change topsoil organic matter. But Greene (1935), McKee (1982), Metz and others (1961), and Wahlenberg (1935) found slight increases through downward leaching. Increases are greater for periodic than annual burns and winter than summer burns. Biennial underburns in Georgia flatwoods increased topsoil organic matter by 25 percent (McKee and Lewis 1982). But 42 years of annual underburns in Florida reduced soil organic matter by 61 tons per acre (Barnette and Hester 1930), and Murad and others (1979) measured reductions of 25 percent in Louisiana.

Conclusions

Light slash burns may consume 25 percent of litter and duff but slightly increase topsoil organic matter. Moderate burns may consume 50 percent of litter and duff; topsoil organic matter may initially decrease by 5-10 percent, but this loss might be made up by leaching inputs. Severe burns may consume 90 percent of litter and duff and reduce topsoil organic matter by 50 percent.

Annual underburns may reduce litter and duff by 70 percent and increase soil organic matter by 5 percent, for a net reduction of 15-20 percent. Underburns every 3-4 years may reduce litter and duff by 50 percent but increase soil organic matter by 15 percent, for a net reduction of 5 percent. Underburns every 5-7 years may reduce litter and duff by 30 percent but increase topsoil organic matter by 20 percent, for a net increase of 5 percent.

Severe slash burns cause major reductions in total organic matter that require years to replace. Underburns more frequent than every 3 years may reduce total organic matter by at least 10 percent. On poor sites low in organic matter, moderate slash burns and underburns more frequent than every 5 years may prevent long term site recovery.

EFFECTS ON NITROGEN

Nitrogen (N) limits growth on most forest sites in the South (Fox and others 1986; Wells and Morris 1982). Most N is in vegetation, forest floor, and soil organic matter that has developed over centuries (Pritchett and Wells 1978). Soil N is concentrated in topsoil, decreasing rapidly with depth. McKee (1982) found half of N in the top 16-20 inches of soil to be in the top 6 inches.

Not all N is available to plants. Most is tied up in organic compounds, so microbes must mineralize it into inorganic ammonium and nitrate to make it available (Jorgensen and Wells 1986). Soil N mineralizes too slowly to supply nutritional demands of young stands (Van Lear and others 1983), so forest floor N and its mineralization rate strongly affect growth (Neary and others 1984; Vitousek and Matson 1985a; Wells and Morris 1982).

Effects of fire on N depend on size of N pool and on losses and inputs to it. Losses from burning occur via volatilization, ash convection, leaching, and erosion. Inputs occur by precipitation and fixation.

Nitrogen Pool

N pools have been estimated for mature oak-hickory in the Blue Ridge Mountains (Swank and Waide 1980) and for loblolly pine at ages 16 (Jorgensen and Wells 1986) and 22 (Tew and others 1986) in the Piedmont. These N pools (lb/ac) are:

	Mature Oak-Hickory	16-Year Loblolly	22-Year Loblolly
Leaves	87	73	48
Branches	103	54	40
Stems	173	102	101
Roots	135	57	50*
Forest Floor	122	274	320
Soil	3631	1564	3512
TOTAL	4251	2124	4071

* Assumed because value not given in Tew and others (1986)

The mature oak-hickory stand had more N in vegetation and less N in forest floor than the two young pine stands. Hardwoods are generally more nutrient demanding than pines and N in pine litter decomposes slowly (Jorgensen and Wells 1986).

New stands quickly amass nutrients and develop forest floor at the expense of soil. The forest floor builds up quickly between stand closure (age 5) and maximum foliage (age 20), holding N in reserve for later cycling while trees get most of their nutrients from soil by microbial mineralization. After age 20, soil N changes little as most N is provided by forest floor decomposition. Accumulation of N in trees and forest floor is rapid through age 20, slows through age 35-40, and reaches equilibrium thereafter. Residual soil N becomes available at only 3-4 percent per year (Hough 1982; Jorgensen and Wells 1986; Jorgensen and others 1975). Understory vegetation contains only a tiny fraction of total N pool (Neary and others 1984).

Switzer and others (1968) modeled N accumulation in loblolly pine stands through age 60. Based on their work and that of Swank and Waide (1980) and Jorgensen and Wells (1986), the following values of N (lb/ac) are considered typical:

	Mature Hardwoods	Mature Pines
Leaves	90	40
Branches	100	70
Stems	170	140
Roots	140	60
Forest Floor	230	400

Soil N can vary greatly depending on climate, soil type, and erosion history. Swank and Waide (1980) reported soil N of 4190-6070 lb/ac for Appalachian coves. In the Coastal Plain, McKee (1982) found 3430-4010 lb/ac in Louisiana rolling uplands, 2640-3300 lb/ac in South Carolina flatwoods, and 1610-1870 lb/ac in Alabama upper hills. Values for Florida sands are 1,010 (Morris and Pritchett 1982), 1,000-2,000 (Hollis and others 1978), and 1960-2500 (McKee 1982) lb/ac. In the Piedmont, Jorgensen and Wells (1986) and Van Lear and others (1983) found 1560-2080 lb/ac for medium and good sites, and Fox and others (1986) found 570-1040 lb/ac in depleted soils. Based on these findings, N pools (lb/ac) considered typical for residual soils (0-12 inches) and total site are:

Soil Type*	Soil Only	Total Site
Poor soils	800	1500
Fair soils	1800	2500
Good soils	2800	3500

* Poor = severely eroded, entisols; fair = inceptisols, spodosols, partly eroded; good = other residual soils.

Nitrogen Loss from Slash Burns

Slash burns can release much N from slash, forest floor, and topsoil. Loss is directly related to fire severity (Pritchett 1976). A moderate to severe slash burn in the Pacific Northwest released 669 lb/ac (Youngberg and Wollum 1976), while a light slash burn in the Florida flatwoods released 230 lb/ac from litter, duff, and topsoil (Burger and Pritchett 1988). Prescribed fire reduced site N by 10 percent in chaparral by consuming vegetation and litter and volatilizing 10 percent of N in the top 0.8 inch of soil (DeBano and Conrad 1978).

N Loss from Slash

N is lost from slash by volatilization and ash convection. Losses of 48 percent for a moderate burn and 80-96 percent for severe burns have been cited (Campbell and others 1977; Grier 1975, 1982). N loss has varied with slash consumption as follows: slash 79 percent, N 87 percent (Powers 1976); slash 27 and 51 percent, N 32 and 74 percent (Feller and others 1983); slash 75-80 percent, N 86-88 percent (Klemmedson 1976); slash 65-76 percent, N 66-76 percent (Little and Klock 1985). Tew and others (1986) assumed that a light slash burn volatilized 50 percent of N in charred areas (41 lb/ac). Moderate burns in the Appalachians have consumed about 50 percent of slash (Sanders and Van Lear 1988).

N lost from slash is about proportional to fraction of slash consumed. Typical fractions of slash and N lost are 25 percent for light burns, 50 percent for moderate burns, and 90 percent for severe burns. Assuming slash to consist chiefly of leaves and branches, N loss from slash (lb/acre) is typically:

	Hardwood	Pine
Light Burn	48	28
Moderate Burn	95	55
Severe Burn	171	99

N Loss from Forest Floor

N is lost from forest floor by volatilization, ash convection, and leaching. Up to 50 percent may be lost in light to moderate burns (Feller 1982). Barnett (1984) assumes forest floor consumption of 50 percent for moderate burns and 90 percent for severe burns. N loss has varied with forest floor consumption as follows: forest floor 23 and 32 percent, N 20 and 38 percent (Feller and others 1983); forest floor 75-80 percent, N 86-88 percent (Klemmedson 1976); forest floor 3 percent, N 5 percent (Stednick and others 1982); forest floor 25 percent, N 94 lb/ac (Jurgensen and others 1981); forest floor 31-58 percent, N 20-53 percent (Little and Klock 1985); forest floor 31 percent, N 24 percent (DeByle 1980). In the Piedmont, light burns have not decreased forest floor N or have volatilized only 41 lb/ac (Fox and others 1986; Tew and others 1986).

N lost from forest floor is about proportional to fraction of forest floor consumed (Kodama and Van Lear 1980). Typical fractions of forest floor and N lost are 25 percent for light burns, 50 percent for moderate burns, and 90 percent for severe burns. Typical N loss from forest floor (lb/ac) is:

	Hardwood	Pine
Light Burn	58	100
Moderate Burn	115	200
Severe Burn	207	360

N Loss from Soil

N is lost from soil by volatilization, leaching, and erosion. Lab tests show loss from organic matter by volatilization to begin at 200 C (14 percent) and be 40-50 percent at 300 C, 67 percent at 500-700 C, and 75 percent at 800-825 C (Grier 1975; Knight 1966; White and others 1973).

In the Pacific Northwest, a severe slash burn might volatilize 180-540 lb/ac, depending on N content of topsoil (Barnett 1984). A Florida burn reduced forest floor and topsoil N from 1140 to 910 lb/ac (Burger and Pritchett 1984). But a light burn in the Virginia Piedmont increased topsoil N (Fox and others 1986), and light to moderate burns in Oregon and Texas did not change topsoil N (Little and Klock 1985; Pehl 1984).

Using the above volatilization data and typical soil temperatures, potential N volatilized from soil (percent) is:

	0-1 in	0-2 in
Light Burn	0	0
Moderate Burn	15	0
Severe Burn	50	5

Leaching loss in stormflow and deep seepage is 0.2-2.7 lb/ac/yr for undisturbed forest (Blackburn and others 1985; Duffy 1985; Fox and others 1986). Harvest and burning increase leaching via increased streamflow and nitrogen mobility. Light burns increased N leaching for one year by 0.6 lb/ac in east Texas and 1.4 lb/ac in the Virginia Piedmont. A severe slash burn in Oregon increased N leaching by 10 lb/ac/yr for at least two years (Brown and others 1973). Typical leaching loss for undisturbed forest is 1 lb/ac/yr. Typical leaching losses from harvest and slash burning are 1 lb/ac for light burns, 3 lb/ac for moderate burns, and 20 lb/ac for severe burns. Erosion losses are discussed later.

N Recovery after Slash Burns

N is input to a site by atmospheric input and fixation. Atmospheric input has averaged 5.5 lb/ac/yr in the South Carolina Piedmont and 8.2 lb/ac/yr in Mississippi and Tennessee (Duffy 1985; Van Lear and others 1983). Jorgensen and Wells (1986) suggest an average of 5.4 lb/ac/yr over loblolly pine's range. It is assumed here that atmospheric inputs average 5 lb/ac/yr.

Nonsymbiotic N fixation by bacteria and algae can be significant after burning due to stimulation of such organisms in warm, moist soil. Fixation can be as low as 0.05 lb/ac/yr in undisturbed forest, and may increase to 1-12 lb/ac/yr after burning depending on organic matter and soil chemistry (Jorgensen and Wells 1971). Fixation in the South Carolina Piedmont was 3.3 lb/ac/yr in uncut stands and 2.1 lb/ac/yr after clearcutting due to lower topsoil moisture, with recovery taking 5 years (Van Lear and others 1983). Slash burns in the Pacific Northwest increased available N but decreased fixation (Jurgensen and others 1981). Thirty years of annual and 4-year underburns in Tennessee to not affect nonsymbiotic fixation (Vance and others 1983). Added nonsymbiotic fixation following slash burning is assumed here to be 20 lb/ac.

Symbiotic N fixation by plants can be significant after burning (Jorgensen and Wells 1986; Van Lear and others 1983). N-fixing plants in the East have rather small biomass and fixation rates (Wells and others 1979), but legumes can be 5 times as abundant after underburns (Cushwa and others 1969). After a fire, plants may fix up to 100 lb/ac depending on plant density and growth environment (Jorgensen and Wells 1986). Symbiotic N fixation after slash burns has not been measured, but legumes are common on burned areas (Jorgensen and Wells 1986; Van Lear and others 1983). Symbiotic fixation is assumed here to total 100 lb/ac after a slash burn.

Available Nitrogen and Nitrogen Mineralization

Available N limits plant growth and may be changed by fire. Mineralized N is the portion of soil N that becomes available. In the Virginia Piedmont, harvest and slash burning increased N mineralization from 34 to 52 lb/ac for 1 year (Fox and others 1986). But a Florida slash burn reduced N mineralization (Burger and Pritchett 1984), and annual underburns in South Carolina flatwoods decreased N uptake (Wells 1971). Repeated annual underburns in Tennessee decreased topsoil ammonium by 44 percent and reduced N mineralization by 31 percent. Underburns every 4 years decreased ammonium and N mineralization by 30 and 17 percent, respectively. Changes in N-containing organic matter were thought to make it less susceptible to microbial mineralization (Vance and Henderson 1984).

Slash burns and underburns may decrease (Bell 1987) or increase available soil N (Covington and Sackett 1984; Jorgensen and Wells 1971; Jurgensen and others 1981; McKevlin and McKee 1986; Mroz and others 1980; Phillips and Goh 1985; Ryan and Covington 1986; Schock and Binkley 1986; Vitousek and Matson 1985b; Wilbur and Christensen 1983). Increases are less after more severe fires (Vitousek and Matson 1985b) and often last a year or less (Jurgensen and others 1981; Mroz and others 1980; Ryan and Covington 1986; Vitousek and Matson 1985b).

Fires usually increase available soil N and enhance revegetation. The increase is short-lived, however, and N mineralization may decrease. N pool is vital to soil quality (Wells and Morris 1982), and so is used here to reflect long term soil productivity. In fact, since forest floor and topsoil are where most losses occur and are most crucial to biomass production, use of total N pool may underestimate adverse effects on soil (Feller and Kimmins 1984).

Nitrogen Budgets

N budgets give some idea of harvest and slash burning effects on long term N pool. Typical N budgets (lb/ac) for pine stands on 60-year rotations are:

	Unburned	Light	Moderate	Severe
LOSSES				
Harvest (stem only)	140	140	140	140
Burn: Slash	0	28	55	99
Forest Floor	0	100	200	360
Soil	0	0	60-105	220-385
Leaching	60	61	63	80
Erosion	0	0	1	11
TOTAL	**200**	**329**	**519-564**	**910-1075**
INPUTS				
Atmospheric	300	300	300	300
Nonsymbiotic Fixation	180	200	200	200
Symbiotic Fixation	20	100	100	100
TOTAL	**500**	**600**	**600**	**600**
NET BUDGET				
Depleted Soils	**+300**	**+271**	**+81**	**-310**
Poor Soils	**+300**	**+271**	**+60**	**-387**
Rich Soils	**+300**	**+271**	**+36**	**-475**

Severe slash burns cause major long-term N loss. Losses after one rotation are 21 percent of site total on poor soils, 16 percent on fair soils, and 14 percent on good soils. N loss of 300 lb/ac on poor soils might reduce timber yields by 25 percent or more (Neary and others 1984). Moderate slash burns should prevent long-term recovery of poor soils.

Nitrogen Loss from Underburns

N is lost from underburns mostly by forest floor consumption. Volatilization of soil N is minor. It is commonly believed that underburns reduce forest floor N by volatilization and increase topsoil N by leaching, with minor net effects on total site N (Jorgensen and Wells 1986; Wells 1971).

In South Carolina flatwoods, a winter burn volatilized 100 of 300 lb/ac of forest floor N (Wells 1971). Lighter burns released 10-36 lb/ac (Richter and others 1982). Burns every 1-2 years in palmetto-gallberry consumed 6,200 lb/ac of understory and forest floor and volatilized 37 lb/ac N; 5-year burns consumed 16,700 lb/ac and volatilized 100 lb/ac N; 8-year burns consumed 22,600 lb/ac and volatilized 173 lb/ac N (Hough 1981). In longleaf-wiregrass, a winter underburn volatilized 70 percent of forest floor N but did not change soil N; a reburn the next year did reduce soil N (Christensen 1977). In the Piedmont, a summer burn that consumed 19 percent of forest floor reduced its N by 13 percent (Kodama and Van Lear 1980). N lost from three annual underburns was 84-143 lb/ac (Van Lear and others 1983).

Early studies suggested that repeated underburns slightly increase topsoil N (Greene 1935; Heyward 1936; Wahlenberg 1935). Long-term effects on site N depend on fire frequency and season. Repeated annual burns in Florida reduced soil N by 1,130 lb/ac (Barnette and Hester 1930). Annual summer burns in Arkansas loess reduced topsoil N by 19 percent, while biennial burns showed no significant effect (Moehring and others 1966). Biennial winter burns in Georgia flatwoods decreased forest floor N from 442 to 75 lb/ac and increased topsoil N from 240 to 310 lb/ac, for a net loss of 297 lb/ac (McKee and Lewis 1983). Forest floor N in four Coastal Plain sites decreased by 59-95 percent for annual burns and 26-32 percent for 7-year burns (McKee 1982), and topsoil N changed in South Carolina flatwoods as follows:

7-year winter burns: +30 lb/ac
Annual winter burns: +122 lb/ac

7-year summer burns: -114 lb/ac
Annual summer burns: -323 lb/ac

Typical long-term loss of forest floor N is 70 percent for annual burns, 50 percent for 3-4 year burns, and 30 percent for 5-7 year burns. Our forest floor N contents and McKee's (1982) findings of soil effects yield typical N budgets (lb/ac) for good soils of:

	ANNUAL		3-4 YEAR		5-7 YEAR	
	Summer	Winter	Summer	Winter	Summer	Winter
Forest Floor	-280	-280	-200	-200	-120	-120
Soil	-320	+120	-220	+ 80	-120	+ 30
TOTAL	-600	-160	-420	-120	-240	- 90

Using these values and adjusting for lower N content of poor and fair soils, typical percent reductions in total site N for underburns would be:

	ANNUAL		3-4 YEAR		5-7 YEAR	
	Summer	Winter	Summer	Winter	Summer	Winter
Poor soils	31	11	22	8	13	6
Fair soils	21	6	15	5	8	4
Good soils	17	5	12	3	7	3

Summer and annual burns, and burns in poor soils, pose significant risks to soil quality. In South Carolina flatwoods, summer and annual winter underburns slightly decreased diameter growth, and annual burns slightly decreased height growth, of mature pines from age 40 to 80. Changes were not significant because the stands were too old to respond (Waldrop and others 1987). In Alabama rolling uplands 2-year winter, spring, and summer burns under longleaf pine reduced height growth by 10 percent, diameter growth by 13 percent, and volume growth by 21 percent from age 14 to 24. Identical burning regimes from age 50 to 60 reduced diameter growth by 11 percent (Boyer 1987). It was speculated that longer burning cycles might not reduce growth.

Nitrogen Loss from Grassland Burns

Most biomass and nutrients in grasslands are below ground. N loss is a small fraction of N pool and is much less significant than in forest, and high cation exchange capacities prevent significant leaching. By removing excess litter and creating temperatures favoring nitrification, fire generally increases grassland productivity (Boerner 1982). Winter burns in Texas prairie with maximum surface temperatures of 400-450 C reduced mulch loads by more than 95 percent but did not affect soil N concentrations (Mutz and others 1985). Periodic burning increased productivity of tallgrass prairie by creating temperatures favoring plant growth and N mineralization, despite loss of 15 percent of site N pool (Ojima and others 1985). But burning for 2 consecutive years reduced grass production by 42 percent in central Louisiana (Cassady 1953).

EFFECTS ON OTHER NUTRIENTS

Studies of effects of prescribed fire on phosphorus (P), potassium (K), calcium (Ca), and magnesium (Mg) are limited. Prescribed fire causes some of these nutrients to be lost to the atmosphere by ash convection and some to be leached into the topsoil (Jorgensen and Wells 1986). At extreme temperatures, some can be vaporized into the atmosphere (Raison and others 1985).

Slash burns in the West have increased pH by up to 1.2 units in 0-3 inches soil (Isaac and Hopkins 1937). Light slash burns have temporarily increased pH and total and available P, K, Ca, and Mg in topsoil (DeByle 1980; Jurgensen and others 1981; Van Lear and Danielovich 1988). Severe burns released 60 percent of P to the atmosphere, but K, Ca, and Mg were later leached into the soil (Grier 1973). A severe burn in jack pine increased pH for more than 15 months; P, K, Ca, and Mg were initially increased and then leached through the sandy soil (Smith 1970). One winter burn in east Texas increased pH but did not affect P, K, Ca, or Mg (Pehl 1984), while another increased P and Ca but did not affect pH, K, or Mg (Stransky and others 1983). In the Piedmont, Tew and others (1986) computed no net loss of P, K, Ca, or Mg from sites burned lightly.

In Arkansas loess, 9 annual underburns increased P in topsoil but did not affect pH, K, Ca, or Mg (Moehring and others 1966). In Tennessee, underburns increased soil P (Bruyn 1984). A winter burn in longleaf-wiregrass increased available soil P, K, Ca, and Mg for less than 6 months, while a reburn the next year reduced them (Christensen 1977). A spring underburn in Louisiana increased pH and available P, K, Ca, and Mg in 0-6 inch soil, with return to pre-burn levels 1 year later (Linnartz and others 1984). In Georgia flatwoods, an underburn increased these nutrients in topsoil for less than 2 years (Pehl and others 1986). But in Georgia rolling uplands, periodic winter burns did not change availability of these nutrients in topsoil (Suman and Carter 1954). In five Coastal Plain sites, repeated burns reduced forest floor nutrients by up to 88 percent for P, 82 percent for K, 92 percent for Ca, and 83 percent for Mg. Available P and K and total Ca and Mg were increased in topsoil (McKee 1982; McKee and Lewis 1983). Concentrations of phosphate, K, and Mg were increased in 0-2 inch peat by an underburn in a North Carolina pocosin (Wilbur and Christensen 1983).

Piedmont underburns reduced forest floor P by 8 percent, K by 20 percent, Ca by 9 percent, and Mg by 22 percent (Kodama and Van Lear 1980). Losses of K and Ca from eroded watersheds exceeded inputs over a rotation. But losses due to 3 annual burns were only 2-4 percent of total losses, most of which occurred via leaching and stormflow (Van Lear and others 1985).

In general, slash burns and underburns do not appear to impair soil quality via effects on P, K, Ca, Mg, or pH. In fact, underburns accelerate P cycling and may improve quality of soils deficient in P (McKevlin and McKee 1986). Leaching of Ca and Mg and consumption of organic acids increase topsoil pH by 0.1-0.9 units (Binkley 1986; Heyward 1936; McKee 1982; Wahlenberg 1935; Wells 1971). These effects slow soil weathering.

EFFECTS OF PRESCRIBED FIRE ON RUNOFF, EROSION, AND SEDIMENT

Effects of prescribed fire on runoff, erosion, and sediment depend on its effect on ground cover and plant water use. Moderate slash burns that retain ground cover but kill most plants should produce small increases in stormflows and channel sediment and negligible increases in surface runoff and erosion. Severe slash burns that expose much bare soil should induce surface runoff and erosion and produce larger increases in stormflows and channel erosion (Tiedemann and others 1979; Van Lear and Waldrop 1987).

Effects of slash burns have been little studied in the East. Western studies have found increases in stormflow and sediment yield related to fire severity, soil type, and topography (Tiedemann and others 1979). A slash burn in east Texas exposed 11 percent mineral soil for one year with streams protected by 20-66 foot buffers. Stormflows increased the first year only, and sediment yield did not increase (Blackburn and others 1986). A slash burn followed by contour ripping in the Ouachita Mountains exposed 57 percent mineral soil with ephemeral streams unprotected. Stormflows did not increase, but sediment yield increased 680 percent in year one, 340 percent in year two, and 200 percent in year three (Miller 1984). In the Blue Ridge Mountains, an intense slash burn done when duff and large fuels were wet exposed less than 15 percent mineral soil, retained a fine root mat in topsoil, and did not change infiltration or erosion (Van Lear and Danielovich 1988).

Underburns have been more studied. Early runoff-erosion studies showed annual underburns to significantly increase erosion from 0.01-0.05 to 0.11-0.36 ton/ac/yr (Ralston and Hatchell 1971). But Piedmont underburns that retained effective ground cover produced little or no erosion, with significant erosion confined mostly to firelines (Brender and Cooper 1968; Cushwa 1971; Goebel and others 1967).

Ursic (1969) measured runoff and sediment yield from two 2-3 acre ephemeral, grass-covered watersheds in loess uplands that were winter burned and planted to pine. On one watershed, bare soil was increased from 0 to 25 percent by burning and to 43 percent the next summer by litter decomposition; first-year increases were 47 percent for mean peak flow and 18-fold for sediment yield. On the other watershed, bare soil increased from 16 to 42 and then 52 percent; first-year increases were 3-fold for mean peak flow and 78-fold for sediment yield, and second-year sediment increase was 7-fold. Most of the sediment increase in this second watershed came from a small reactivated gully. Loss of transpiring vegetation caused part of the peak flow increase, which in turn caused part of the sediment increase via channel erosion.

Ursic (1970) studied two other 2-3 acre ephemeral, hardwood-covered watersheds that were winter burned, herbicide treated, and planted to pine. On the sandy loam watershed, bare soil increased from 0 to 17 percent due to burning and declined to 8 percent in three years; three-year increases were 53, 31, and 13 percent for mean peak flow and 48, 8, and 0 percent for sediment yield. On the loess watershed, bare soil increased from 2 to 11 percent and then declined to 2 percent; increases were 33, 15, and 34 percent for mean peak flow and 119, 127, and 121 percent for sediment yield. Burning increased bare soil by less than 20 percent, so the peak flow increase was caused by hardwood removal. The sediment occurred from channel erosion caused by higher peak flows.

Underburns in pine-grass watersheds reduced infiltration and increased sediment yield for 10 months (Dobrowolski and others 1984). Burns in dense, herbicide-treated oak oak savannah reduced infiltration for 6 months and increased simulated erosion for less than 1 year (Lloyd-Reilley and others 1984). Two successive underburns in 1.5-3.1 acre ephemeral watersheds in the South Carolina Piedmont consumed only one-third of the forest floor, exposed less than 1 percent mineral soil, and did not significantly increase runoff or sediment yield. Sediment increases after a third underburn followed by a clearcut were attributed to increased flows from harvest (Douglass and Van Lear 1983; Van Lear and others 1985).

Effects on Stormflows and Channel Erosion

Timber harvest increases stormflow volumes and peaks in proportion to percent of stems cut. Increases from clearcuts in 1-3 acre ephemeral watersheds average 40 percent and last 1 year (Douglass and others 1983; Settergren and Krtansky 1987; Ursic 1970). Added increases are typically negligible for light slash burns and 40 percent for moderate slash burns. Both burns expose less than 20 percent bare soil and retain the fine root mat in topsoil. The small increases from moderate burns are due mostly to reduced interception and evapotranspiration by vegetation. Severe slash burns cause greater increases by exposing much soil and promoting surface runoff. Typical increases are 200 percent, and recovery may take 3 years.

Underburns do not affect plant water use because the overstory is retained. Grassland burns reduce plant water use for only a few days or weeks until grass regrows. These burns do not significantly affect stormflows.

Existing studies where surface erosion was minimal suggest that channel erosion increases roughly in proportion to peak flow (Blackburn and others 1986; Ursic 1970). Typical increases in channel erosion should be on the order of 200 percent for severe slash burns, 40 percent for moderate slash burns, and negligible for light slash burns, underburns, and grassland burns.

Effects on Surface Erosion and Sediment

Potential surface erosion is estimated by the Universal Soil Loss Equation (USLE). It depends on site characteristics (climate, soil, topography) and degree of surface disturbance (Dissmeyer and Cost 1984). Dissmeyer and Foster (1984) presented a method for using USLE on forest lands. Its equation is:

$$A = RKLSCP$$

Where: A = computed soil loss (tons/ac/yr);

R = rainfall-runoff factor in erosion index (EI) units -- one EI unit = 100 (foot-tons/ac)(inches/hr);

K = soil erodibility factor (soil loss rate per EI unit on a 72.6 foot, 9 percent slope continuously in clean-tilled fallow);

L = slope length factor (ratio of soil loss from site to that from a 72.6 foot length);

S = slope steepness factor (ratio of soil loss from site to that from a 9 percent slope);

C = cover-management factor (ratio of soil loss from site to that from one in tilled, continuous fallow);

P = practice factor (ratio of soil loss with contour tilling to that with straight-row tilling up and down the slope).

Typical values of R, K, L, and S were derived for landtypes in the South from Dissmeyer and Stump (1978), as modified by national forest data (Table 1). Typical CP factors are 0.000 for light burns, 0.002 for moderate burns, and 0.015 for severe burns. Using these USLE factors enables potential surface erosion for the entire recovery period to be computed (Table 2).

N content of eroded soil is no more than 0.2 percent (Hollis and others 1984). Using this value, average risks to productivity from erosion loss of N are minimal (0-5 percent of site total) for slash burns on all soils in all landtypes. Risks may be higher for severe burns on steep, erodible sites. Effects of erosion on soil productivity are rather minor compared to heating loss of organic matter and N. The major impact of erosion is on water quality.

The portion of eroded soil delivered to streams is the sediment delivery ratio (SDR). Roehl (1962) related watershed size to percent of erosion reaching a downstream point. This relation reflects that (1) most sediment is produced near the

Table B-1.--USLE factors for southern landtypes

	Slope (%)	Length (feet)	--- USLE Factors ---		
			LS	R	K
COASTAL PLAIN					
Oak Savannahs	5	200	0.76	275	0.24
Upper Hills	10	180	1.85	275	0.24
Rolling Uplands	5	100	0.54	425	0.24
Clay Flatlands	3	80	0.27	360	0.32
Loess Uplands	10	110	1.40	350	0.37
FL Sand Ridges	3	200	0.35	375	0.17
Miss. Valley	1	100	0.13	350	0.37
Gulf Flatwoods	1	200	0.16	500	0.20
Atlantic Flatwoods	1	200	0.16	350	0.24
PIEDMONT	15	110	2.70	260	0.32
BLUE RIDGE					
Narrow Ridge	35	100	10.26	160	0.24
Blue Ridge Mtns.	40	100	12.70	250	0.24
Unaka Mtns.	40	100	12.70	200	0.24
RIDGE AND VALLEY					
Folded Highlands	30	120	8.75	150	0.28
Faulted Lowlands	25	120	6.48	320	0.28
APPALACHIAN PLATEAUS					
Cumberland Mtns.	30	160	10.10	150	0.28
Kentucky Basin	25	160	7.49	200	0.28
Table Plateaus	15	110	2.70	320	0.28
OZARK PLATEAUS					
Springfield Plateau	20	180	5.49	260	0.28
Boston Mtns.	20	150	5.02	280	0.32
OUACHITA					
Arkansas Valley	15	150	3.15	300	0.32
Ouachita Mtns.	20	100	4.10	320	0.32

Table B-2.--Potential erosion (tons/ac) for recovery period in Southern landtypes

	Undisturbed	Light	Moderate	Severe
COASTAL PLAIN				
Oak Savannahs	0.01	0.01	0.10	0.75
Upper Hills	0.00	0.00	0.24	1.83
Rolling Uplands	0.00	0.00	0.11	0.83
Clay Flatlands	0.00	0.00	0.06	0.47
Loess Uplands	0.00	0.00	0.36	2.72
FL Sand Ridges	0.00	0.00	0.04	0.33
Miss. Valley	0.00	0.00	0.03	0.25
Gulf Flatwoods	0.00	0.00	0.03	0.24
Atlantic Flatwoods	0.00	0.00	0.03	0.20
PIEDMONT	0.00	0.00	0.45	3.37
BLUE RIDGE				
Narrow Ridge	0.00	0.00	0.79	5.91
Blue Ridge Mtns.	0.00	0.00	1.52	11.43
Unaka Mtns.	0.00	0.00	1.22	9.14
RIDGE AND VALLEY				
Folded Highlands	0.00	0.00	0.74	5.51
Faulted Lowlands	0.00	0.00	1.16	8.71
APPALACHIAN PLATEAUS				
Cumberland Mtns.	0.00	0.00	0.84	6.29
Kentucky Basin	0.00	0.00	0.85	6.36
Table Plateaus	0.00	0.00	0.48	3.63
OZARK PLATEAUS				
Springfield Plateau	0.00	0.00	0.80	6.00
Boston Mtns.	0.00	0.00	0.90	6.75
OUACHITA				
Arkansas Valley	0.00	0.00	0.60	4.54
Ouachita Mtns.	0.00	0.00	0.84	6.30

headwaters where relief and dissection are greatest, and (2) sediment storage in channels and floodplains increases with watershed size (Schumm 1977). When applied to average sizes of fourth order watersheds measured by national forests for 11 landtypes, this method computed SDR's of 0.23-0.35.

Most national forests occupy headwaters and our concern is the portion of eroded soil reaching any channel. Roehl's method is not suited for us. Soil eroded from ridges and upper slopes rarely reaches a channel. The sediment source zone is a streamside area whose extent varies with slope steepness and dissection.

Drainage density (miles of channel per square mile of watershed) was measured by national forests in 11 landtypes. Average values were 3.4 in Florida sand ridges, 7.4 in Middle Coastal Plain, and 9.9-14.0 in Upper Coastal Plain, Piedmont, and mountains. The higher values are typical of terrain with extreme slopes and rainfall. Channels were drawn on topographic maps by inexperienced people, and many do not have a corresponding active channel on the ground. These suspect values were reduced to more realistic levels of 3.0 for landtypes with 1-3 percent slopes; 6.0 for those with 5 percent slopes; and 10.0 for those with 10+ percent slopes. Roughly half the drainage network is perennial and intermittent streams protected by buffers. The drainage densities of ephemeral streams only are thus 1.5, 3.0, and 5.0.

Swift (1986) found width of sediment source zone (feet) to be 50 + 3 (slope %) for unsurfaced roads with bare fills and 40 + 1.4 (slope %) for surfaced roads with grassed fills. The wider zone includes effects of concentrated flows and mass fill failure. The narrower zone represents sheet-rill erosion processes and so is used in our analysis.

Not all soil eroded from the sediment source zone reaches the channel. Sediment delivery decreases with distance from stream. It is assumed that 95 percent is delivered from the nearest 10 percent of the zone and 5 percent is delivered from the farthest 10 percent, for an average of 50 percent. Typical SDR's are:

Lower Coastal Plain; Clay Flatlands	0.01
Oak Savannahs; Rolling Uplands	0.03
Upper Hills, Loess Uplands	0.05
Piedmont	0.06
Mountains	0.08

Because sediment yields must be assessed for whole watersheds, evaluation of their effects must be deferred to a cumulative effects analysis. Sediment impacts should generally be significant only for severe burns in the Upper Hills, Loess Uplands, Piedmont, and mountains.

EFFECTS ON CHEMICAL WATER QUALITY

Fire generally increases solubility and leaching potential of soil nutrients such as nitrate, ammonium, phosphate, sulfate, Ca, Mg, K, Na, chloride (Cl), and bicarbonate. Removing ground cover increases potential for erosion loss of these nutrients (Tiedemann and others 1979).

Effects of Slash Burns

Tiedemann and others (1979) summarized results of western slash burns on stream nutrients. Even severe burns with no buffers did not increase nitrate to more than 7.6 ppm. Ammonium is increased for less than a month after a fire, but severe burns did exceed 0.5 ppm. Organic-N also increased by up to 100 percent by delivery of organic matter to streams. Even severe burns do not affect phosphate concentrations significantly. Results on cations are variable, but suggest that increases are minor and do not impair water quality.

A slash burn covering 19 percent of a watershed in British Columbia produced maximum stream concentrations of 5.4 (K), 1.9 (Na), 1.4 (Mg), and 12.6 (Ca) ppm on the day of burning (Feller and Kimmins 1984). Nitrate achieved a maximum of 2.5 ppm, but returned to pretreatment levels within two years.

A clearcut followed by chopping and burning in east Texas increased bare soil to 15 percent for 1 year. Nitrate loss did not significantly increase. Total N loss increased significantly for 2 years, but never exceeded 0.6 lb/ac/yr. Orthophosphate and total P losses did not increase significantly (Blackburn and others 1985). In the Virginia Piedmont, a light burn significantly increased mean instream ammonium concentrations to 0.26 ppm. Nitrate increased to 0.61 ppm but not significantly (Fox and others 1986).

The only eastern study to approach a severe slash burn was of a wildfire that burned a tornado-damaged area in the Appalachians and removed 90 percent of the ground cover (Neary and Currier 1982). Mean concentrations (ppm) increased significantly for nitrate (to 0.05), Na (to 1.36), K (to 0.70), Ca (to 0.97), and Mg (to 0.44), but not for ammonium or phosphate. Peak nitrate concentration was only 0.25 ppm and was raised by aerial fertilization of ammonium nitrate on 39 percent of the watershed.

Light to moderate slash burns that retain effective ground cover produce minimal increases in stream nutrient concentrations. Even severe slash burns should not impair water quality, because increases in nutrient concentrations are minor.

Effects of Underburns

An underburn in South Carolina sand hills increased solubility of cations in forest floor but not export in runoff or shallow ground water, except for Na. Neither solubility nor export of nitrate or phosphate increased (Lewis 1974). Burning a grass-covered watershed in the Ouachita Mountains 6 years after it was clearcut did not increase any stream nutrients (Lawson and Hileman 1983).

Underburns in South Carolina flatwoods did not affect ground water chemistry. In addition, 6 underburns that covered 60 percent of a watershed, consumed less than one-third of the forest floor, and were separated from streams by 66 foot buffers did not significantly change stream concentrations of total N, ammonium, nitrate, phosphate, sulfate, Cl, Ca, Mg, K, Na, bicarbonate, H, or specific conductance (Richter and others 1982).

Two succesive underburns in 1.5-3.1 acre ephemeral watersheds in the South Carolina Piedmont consumed only one-third of the forest floor, exposed less than 1 percent mineral soil, and did not increase stream concentrations or export of nitrate, ammonium, phosphate, Ca, Mg, K, or Na (Douglass and Van Lear 1983). Underburns increase solubility and export of some nutrients. Increases in stream concentrations, however, are not significant.

SUMMARY OF EFFECTS

Slash burns occur once per rotation. Risks of adverse effects on soil and water depend on severity of burn and type of site. Underburns and grassland burns occur every 1-7 years. Risks depend more on frequency and season of burn.

Slash Burns

Light slash burns pose no risks to soil or water quality.

Moderate slash burns pose no risks to soil or water quality on most sites. On poor soils, however, they prevent buildup of organic matter and N vital to long term site recovery.

Severe slash burns pose risks to soil quality on all sites via loss of soil biota, structure, and organic matter. Further risks via N loss are extreme on poor soils, high on fair soils, and medium on good soils. Risks of sediment impacts can be significant on steep, erodible sites.

Underburns

Underburns more frequent than every 3 years pose high risks to soil quality on all sites. Soil biota are not affected, but forest floor biota are reduced and cannot fully recover before the next burn. Loss of site organic matter exceeds 10 percent. Annual underburns also impair soil physical properties.

WINTER underburns every 3-4 years pose minimal risks to soil quality on most sites. Forest floor biota fully recover between burns, and soil physics are not impaired. Loss of site organic matter is about 5 percent, and N loss is not a significant portion of site total. On poor soils, however, such underburns prevent buildup of organic matter vital to long term site recovery. Underburns on these soils should thus have a 5-7 year frequency and occur only in winter.

SUMMER underburns every 3-4 years also cause about a 5 percent loss of organic matter. N loss is greater than for winter underburns, possibly by suppression of N-fixing legumes. Risks to soil quality are extreme on poor soils, high on fair soils, and medium on good soils. Risks can be kept low on all but poor soils by alternating winter and summer burns.

Grass Burns

Annual grass burns pose risks to soil quality via reduction of litter biota, damage to soil structure and infiltration capacity, and loss of site organic matter. Risks are negligible for intervals of 3 years or more.

Control of Fire Severity

Severe burns require heat penetration into soil, which depends on duration of heating and soil moisture. Large fuels must burn to produce severe impacts, and soil temperature can't exceed 100 C until all water is evaporated. If burning occurs when large fuels, duff, and soil are moist, severe impacts are unlikely because larger fuels should not burn enough to evaporate all moisture in the topsoil (Martin 1981; Sanders and Van Lear 1988).

An area is considered severely burned only if all litter and duff are consumed and mineral soil is altered on more than 20 percent of the area. Slash burning soon after soaking (0.5 inch or more) rains when duff and large fuels are moist should avoid severe effects. Burns are effective if residual trees are felled, chopped, or herbicide-treated a month before burning to allow curing. Presence of cured fuels increases number of burning days. Burning exhausts root reserves of resprouted hardwoods and enhances effectiveness of treatment (Abercrombie and Sims 1986; Van Lear and Waldrop 1987).

REFERENCES

Abercrombie, J A; Sims, D H. 1986. Fell and burn for low-cost site preparation. Forest Farmer 46(1):14-17.

Agee, J K. 1973. Prescribed fire effects on physical and hydrologic properties of mixed-conifer forest floor and soil. Contrib Rep 143, Water Resour Center, Univ CA, 57 p.

Ahlgren, I F; Ahlgren, C E. 1965. Effect of prescribed burning on soil microorganisms in a Minnesota jack pine forest. Ecology 46:304-310.

Arend, J L. 1941. Infiltration rates of forest soils in the Missouri Ozarks as affected by woods burning and litter removal. J For 39:726-728.

Austin, R C; Baisinger, D H. 1955. Some effects of burning on forest soils of western Oregon and Washington. J For 53(4):275-280.

Auten, J T. 1934. The effect of forest burning and pasturing in the Ozarks on the water absorption of forest soils. USDA For Serv, Cent States For Exp Stn Note 16, 5 p.

Barber, B L; Van Lear, D H. 1984. Weight loss and nutrient dynamics in decomposing woody loblolly pine logging slash. Soil Sci Soc Am Jour 48:906-910.

Barnett, D. 1984. Effects of fire on Coast Range sites. Siuslaw NF, Waldport, OR, 122 p.

Barnette, R M; Hester, J B. 1930. Effect of burning upon the accumulation of organic matter in forest soils. Soil Sci 29:281-284.

Beadle, N C W. 1940. Soil temperatures during forest fires and their effect on the survival of vegetation. J Ecol 28:180-192.

Bell, R L. 1987. Soil nitrogen mineralization and immobilization in response to periodic prescribed fire in a loblolly pine plantation. MS Thesis, Dept For & Env Studies, Duke Univ, 24 p.

Bentley, J R; Fenner, R L. 1958. Soil temperatures during burning related to postfire seedbeds on woodland range. J For 56:737-740.

Binkley, D. 1986. Soil acidity in loblolly pine stands with interval burning. Soil Sci Soc Am J 50:1590-1594.

Blackburn, W H; Wood, J C; DeHaven, M G. 1986. Storm flow and sediment losses from site-prepared forestland in east Texas. Water Resour Res 22(5):776-784.

Blackburn, W H; Wood, J C; DeHaven, M G. 1985. Forest harvesting and site preparation impacts on stormflow and water quality in east Texas. In (Blackmon, B G, ed) Proc, For and Water Qual, a Mid-South Sym, May 1985, Little Rock, AR; p 74-93 [Available from Univ AR, Fayetteville, AR].

Boerner, R E J. 1982. Fire and nutrient cycling in temperate ecosystems. Biosci 32(3):187-192.

Boyer, W. D. 1987. Volume growth loss: a hidden cost of periodic prescribed burning in longleaf pine? S J Appl For 11(3):154-157.

Bower, D R. 1966. Surface soil recovers quickly after burn. USDA For Serv Res Note SO-46, 2p.

Brender, E V; Cooper, R W. 1968. Prescribed burning in Georgia's Piedmont loblolly pine stands. J For 66:31-36.

Brender, E V. McNab, W H; Williams, S. 1976. Fuel accumulations in Piedmont loblolly pine plantations. USDA For Serv Res Note SE-233, 4 p.

Brown, G W; Gahler, A R; Marston, R B. 1973. Nutrient losses after clear-cut logging and slash burning in the Oregon Coast Range. Water Resour Res 9:1450-1453.

Bruyn, P de. 1984. Prescribed fire in a 23-year-old loblolly pine plantation on sloping terrain in west Tennessee. Univ TN, Dep For, Wildlife, & Fish, For Abst 45(7):382-383.

Burger, J A; Pritchett, W L. 1984. Effects of clearfelling and site preparation on nitrogen mineralization in a southern pine stand. Soil Sci Soc Am J 48:1432-1437.

Burger, J. A.; Pritchett, W. L. 1988. Site preparation effects on soil moisture and available nutrients in a pine plantation in the Florida flatwoods. For Sci 34(1):77-87.

Campbell, R E; Baker, M B; Ffolliott, P F. 1977. Wildfire effects on a ponderosa pine ecosystem: an Arizona case study. USDA For Serv Res Pap RM-191, 12 p.

Cassady, J T. 1953. Burning may reduce grass production. USDA For Serv, S For Notes, No 85.

Christensen, N L. 1977. Fire and soil-plant nutrient relations in a pine-wiregrass savannah on the Coastal Plain of North Carolina. Oecologia 31:27-44.

Covington, W W; Sackeett, S S. 1984. The effects of a prescribed burn in southwestern ponderosa pine on organic matter and nutrients in woody debris and forest floor. For Sci 30(1):183-191.

Cromer, R N; Vines, R G. 1966. Soil temperatures under a burning windrow. Aust For Res 2(2):29-34.

Cushwa, C T; Czuhai, E; Cooper, R W; Julian, W H. 1969. Burning clearcut openings in loblolly pine to improve wildlife habitat. GA For Res Counc Res Pap 61, 5 p.

Cushwa, C T; Hopkins, M; McGinnes, B S. 1970. Response of legumes to prescribed burns in loblolly pine stands of the South Carolina Piedmont. USDA For Serv Res Note SE-140, 6 p.

Cushwa, C T; Hopkins, M; McGinnes, B S. 1971. Soil movement in established gullies after a single prescribed burn in the South Carolina Piedmont. USDA For Serv Res Note SE-153, 4 p.

DeBano, L F. 1966. Formation of non-wettable soils--involves heat transfer mechanism. USDA For Serv Res Note PSW-132, 8 p.

DeBano, L F. 1979. Effects of fire on soil properties. Univ CA Div Agr, For Soils Pub 4094, p 109-118.

DeBano, L F. 1981. Water-repellent soils: a state-of-the-art. USDA For Serv Gen Tech Rep PSW-46.

DeBano, L F; Conrad, C E. 1978. The effect of fire on nutrients in a chaparral ecosystem. Ecology 59(3):489-497.

DeBano, L F; Dunn, P H; Conrad, C E. 1977. Fire's effect on physical and chemical properties of chaparral soils. In Proc Envir Conseq of Fire and Fuel Mgt in Medit Ecosys Sym, Palo Alto, CA, Aug 1977; USDA For Serv Gen Tech Rep WO-3:65-74.

DeBano, L F; Rice, R M. 1971. Fire in vegetation management--its effect on soil. In Proc Interdisc Aspects Watershed Mgt Sym, Bozeman MT, Aug 1971:327-346.

DeBano, L F; Rice, R M; Conrad, C E. 1979. Soil heating in chaparral fires: effects on soil properties, plant nutrients, erosion, and runoff. USDA For Serv Res Pap PSW-145, 21 p.

DeBano, L F; Savage, S M; Hamilton, D M. 1976. The transfer of heat and hydrophobic substances during burning. Soil Sci Soc Am Jour 40:779-782.

DeBell, D S; Ralston, C W. 1970. Release of nitrogen by burning light forest fuels. Soil Sci Soc Am Proc 34(6):936-938.

DeByle, N V. 1980. Harvesting and site treatment influences on the nutrient status of lodgepole pine forests in western Wyoming. In Env Conseq of Timber Harv in Rocky Mtn Conif For; USDA For Serv Gen Tech Rep INT-90:137-156.

DeByle, N V. 1981. Clearcutting and fire in the larch/Douglas-fir forests of western Montana--a multifaceted research summary. USDA For Serv Gen Tech Rep INT- 99.

Dissmeyer, G E; Cost, N D. 1984. Multiresource inventories--watershed condition of commercial forest land in South Carolina. USDA For Serv Res Pap SE-247, 26 p.

Dissmeyer, G E; Foster, G R. 1984. A guide for predicting sheet and rill erosion on forest land. USDA For Serv Tech Pub R8-TP-6, 40 p.

Dissmeyer, G E; Stump, R F. 1978. Predicted erosion rates for forest management activities in the Southeast. USDA For Serv Tech Pub R8-TP-?

Dobrowolski, J P; Blackburn, W H; Grelan, H E. 1984. Infiltration rates and water quality of longleaf pine/bluestem range following 22 years of seasonal burning. Soc Range Mgt Ann Mtg 37:13 (Abst only).

Douglass, J E; Van Lear, D H. 1983. Prescribed burning and water quality of ephemeral streams in the Piedmont of South Carolina. For Sci 29(1):181-189.

Duffy, P D. 1985. Nutrient gains and losses for loblolly pine plantations. In Proc For and Water Qual--A Mid-South Sym, Little Rock, AR, May 1985:42-54 [Avail from Univ AR, Dept For Res].

Dunn, P H; Barro, S C; Poth M. 1985. Soil moisture affects survival of microorganisms in heated chaparral soil. Soil Biol & Biochem 17(2):143-148.

Dunn, P H; DeBano, L F. 1977. Fire's effect on the biological properties of chaparral soils. In Int'l Sym on Envir Conseq on Fire and Fuel Mgt in Medit-climate Ecosys (forests and scrublands).

Dyrness, C T; Youngberg, C T. 1957. The effect of logging and slash-burning on soil structure. Soil Sci Soc Am Proc 21:444-447.

Feller, M C. 1982. The ecological effects of slashburning with particular reference to British Columbia--a literature review. Prov BC Ministry of Forests Pub R28-81072.

Feller, M C; Kimmins, J P. 1984. Effects of clearcutting and slash burning on streamwater chemistry and watershed nutrient budgets in southwestern British Columbia. Water Resour Res 20(1):29-40.

Feller, M C; Kimmins, J P; Tsze, K M. 1983. Nutrient losses to the atmosphere during slashburns in southwestern British Columbia. Proc 7th Conf Fire & For Meteor, Am Meteor Soc & Soc Am For, Ft Collins, CO, Apr 25-28, 1983:128-135.

Fonteyn, P J; Stone, M W; Yancy, M A; Baccus, J T. 1984. Interspecific and intraspecific microhabitat temperature variations during a fire. Am Midl Nat 112(2):246-250.

Fox, T R; Burger, J A; Kreh, R E. 1986. Effects of site preparation on nitrogen dynamics in the southern Piedmont. For Ecol and Mgt 15:241-256.

Frandsen, W H; Ryan, K C. 1986. Soil moisture reduces belowground heat flux and soil temperatures under a burning fuel pile. Can Jour For Res 16(2):244-248.

Garren, K H. 1943. Effects of fire on vegetation of the southeastern United States. Bot Rev 9(9):617-654.

Goebel, N B; Brender, E V; Cooper, R W. 1967. Prescribed burning of pine-hardwood stands in the Upper Piedmont of South Carolina. Clemson Univ, SC Agr Exp Stn, For Res Ser 16, 22 p.

Greene, S W. 1935. Effect of annual grass fires on organic matter and other constituents of virgin longleaf pine soils. J Agr Res 50(10):809-822.

Grier, C C. 1973. Effects of fire on the movement and distribution of elements within a forest ecosystem. Dissert Abst, Ecology 33B:154.

Grier, C C. 1975. Wildfire effects on nutrient distribution and leaching in a coniferous ecosystem. Can J For Res 5:599-607.

Grier, C C. 1982. Information presented at Stand Mgt/Fertility Sym, OR State Univ, For Ext Serv and Dept For Sci, Dec 1982.

Harshbarger, T J; Perkins, C J; Martin, R E. 1975. Legume response unrelated to fuel moisture at time of burning. J Range Mgt 28(1):70-71.

Heady, H F. 1956. Changes in a California annual plant community induced by manipulation of natural mulch. Ecology 37:798-812.

Henley, J W; Clarke, E H. 1976. Forest residues--a manageable problem. Soil Cons Soc Am, Land Appl of Waste Materials 1976:98-111.

Heyward, F. 1936. Soil changes associated with forest fires in the longleaf pine region of the South. Am Soil Surv Assoc Bull 17:41-42.

Heyward, F. 1937. The effect of frequent fires on profile development of longleaf pine forest soils. J For 35:23-27.

Heyward, F. 1938. Soil temperatures during forest fires in the longleaf pine region. J For 36:478-491.

Heyward, F; Tissot, A N. 1936. Some changes in the soil fauna associated with forest fires in the longleaf pine region. Ecology 17(4):659-666.

Hollis, C A; Fisher, R F; Pritchett, W L. 1978. Effects of some silvicultural practices on soil-site properties in the lower Coastal Plain. In (C T Youngberg, ed) Forest Soils and Land Use, Proc 5th N Amer For Soils Conf, Ft Collins, CO, CO State Univ; 585-606.

Hosking, J S. 1938. The ignition at low temperatures of the organic matter in soils. J Agr Sci 28:393-400.

Hough, W A. 1981. Impact of prescribed fire on understory and forest floor nutrients. USDA For Serv Res Note SE-303, 4 p.

Hough, W A. 1982. Phytomass and nutrients in the understory and forest floor of slash/longleaf pine stands. For Sci 28(2):359-372.

Humphreys, F R; Lambert, M J. 1965. An examination of a forest site which has exhibited the ash-bed effect. Aust J Soil Res 3:81-94.

Hunt, S M; Simpson, J A. 1985. Effects of low intensity prescribed fire on the growth and nutrition of a slash pine plantation. Aus For Res 15(1):67-77.

Isaac, L A; Hopkins, H G. 1937. The forest soil of the Douglas-fir region and the changes wrought upon it by logging and slash burning. Ecology 18:264-279.

Jorgensen, J R; Hodges, C S. 1971. Effects of prescribed burning on the microbial characteristics of soil. In Proc Presc Burning Sym, Charleston, SC, April 1971, p 107-111 [Avail from SE For Exp Stn, Asheville, NC].

Jorgensen, J R; Wells, C G. 1971. Apparent nitrogen fixation in soil influenced by prescribed burning. Soil Sci Soc Am Proc 35(5):806-810.

Jorgensen, J R; Wells, C G. 1986. Foresters' primer in nutrient cycling. USDA For Serv Gen Tech Rep SE-37, 42 p.

Jorgensen, J R; Wells, C G; Metz, L J. 1975. The nutrient cycle: key to continuous forest production. J For 73:400-403.

Jurgensen, M F; Harvey, A E; Larsen, M J. 1981. Effects of prescribed fire on soil nitrogen levels in a cutover Douglas-fir/western larch forest. USDA For Serv Res Pap INT-275, 6 p.

Kinnes, S S. 1982. Effects of prescribed burning on mycorrhizae of loblolly pine and their fungal associates. PhD Thesis, Dept For & Env Studies, Duke Univ, 176 p.

Kittredge, J. 1938. Comparative infiltration in the forest and open. J For 36:1156-1157.

Klemmedson, J O. 1976. Effect of thinning and slash burning on nitrogen and carbon in ecosystems of young dense ponderosa pine. For Sci 22(1):45-53.

Klemmedson J O; Schultz, A M; Jenny, H; Biswell H H. 1962. Effect of prescribed burning of forest litter on total soil nitrogen. Soil Sci Soc Proc 26(2): 200-202.

Knight, H. 1966. Loss of nitrogen from the forest floor by burning. For Chron 42(2):149-152.

Kodama, H E; Van Lear, D H. 1980. Prescribed burning and nutrient cycling relationships in young loblolly pine plantations. S J App For 4(3):118-121.

Lawson, E R; Hileman, L H. 1983. Nutrient distributions in runoff from Ouachita Mountain watersheds. USDA For Serv Gen Tech Rep SE-24, 6 p.

Lewis, W M. 1974. Effects of fire on nutrient movement in a South Carolina pine forest. Ecology 55:1120-1127.

Lindenmuth, A W; Byram, G M. 1948. Headfires are cooler near the ground than backfires. Fire Control Notes 9(4):8-9.

Linnartz, N E; Hu, Shih-Chang; Langston, H W. 1984. The effects of prescribed burning on some chemical properties of forest soils in southeastern Louisiana. LSU Forestry Note 141, 2 p. LA State Univ Agr Ctr, LA Agr Exp Stn, Baton Rouge, LA.

Little, S N; Klock, G O. 1985. The influence of residue removal and prescribed fire on distributions of forest nutrients. USDA For Serv Res Pap PNW-338, 12 p.

Lloyd-Reilley, J; Scifres, C J; Blackburn, W H. 1984. Hydrologic impacts of brush management with tebuthiuron and prescribed burning on post oak savannah watersheds, Texas. Agric, Ecos, & Env 11:213-224.

Martin, R E. 1981. Prescribed burning techniques to maintain or improve soil productivity. In (Hobbs, S D; Helgerson, O T; eds) Proc Refor of Skeletal Soils, Medford, OR, Nov 1981, p 66-70 [Avail from OR State Univ, For Res Lab, Corvallis, OR].

McKee, W H. 1982. Changes in soil fertility following prescribed burning on Coastal Plain pine sites. USDA For Serv Res Pap SE-234, 23 p.

McKee, W H; Lewis, C E. 1983. Influence of burning and grazing on soil nutrient properties and tree growth on a Georgia Coastal Plain site after 40 years. In USDA For Serv Gen Tech Rep SE-24, Proc 2nd Bien South Silv Res Conf, Atlanta, GA, Nov 1982, p 79-86.

McKevlin, M R; McKee, W H. 1986. Long-term prescribed burning increases nutrient uptake and growth of loblolly pine seedlings. For Ecol & Mgt 17:245-252.

Meginnis, H G. 1935. Effect of cover on surface run-off and erosion in the loessal uplands of Mississippi. USDA Circ 347, 15 p.

Metz, L J; Farrier, M H. 1971. Prescribed burning and soil mesofauna on the Santee Experimental Forest. *In* Proc Presc Burning Sym, Charleston SC, April 1971, p 100-106 [Avail from USDA For Serv, SE For Exp Stn, Asheville, NC].

Metz, L J; Lotti, T; Klawitter, R A. 1961. Some effects of prescribed burning on Coastal Plain forest soil. USDA For Serv, SE For Exp Stn Pap 133, 10 p.

Miller, E L. 1984. Sediment yield and storm flow response to clear-cut harvest and site preparation in the Ouachita Mountains. Water Resour Res 20(4): 471-475.

Moehring, D M; Grano, C X; Bassett, J R. 1966. Properties of forested loess soils after repeated prescribed burns. USDA For Serv Res Note SO-40, 4 p.

Mroz, G D; Jurgensen, M F; Harvey, A E; Larsen, M J. 1980. Effects of fire on nitrogen in forest floor horizons. J Soil Sci Soc Am 44:395-400.

Murad, J L; Berry, C W; Harrison, R E; Malone, W M; Roberts, C S. 1979. Prescribed burning and nematode interaction in the soil of a Southern pine forest. LA Tech Univ, Sch For, Bull No 12, 70 p.

Mutz, J L; Greene, T G; Scifres, C J; Koerth, B H. 1985. Response of Pan American balsamscale, soil, and livestock to prescribed burning. TX Ag Exp Stn Bull B-1492, 14 p.

Neal, J L; Wright, E; Bollen, W B. 1965. Burning Douglas-fir slash: physical, chemical, and microbial effects in the soil. OR State Univ For Res Lab Res Pap 1, 32 p.

Neary, D G; Currier, J B. 1982. Impact of wildfire and watershed restoration on water quality in South Carolina's Blue Ridge Mountains. South J App For 6(2):81-90.

Neary, D G; Morris, L A; Swindel, B F. 1984. Site preparation and nutrient management in southern pine forests. *In* (E L Stone, ed) Proc For Soils and Treatment Impacts, 6th N Amer For Soils Conf, June 1983, Knoxville, TN. Univ TN, Dept For, Wildlife, & Fisheries, Knoxville, TN; p 121-144.

Nissley, S D; Zasoski, R J; Martin, R E. 1980. Nutrient changes after prescribed surface burning of Oregon ponderosa pine stands. Proc 6th Conf Fire & For Meteor, Apr 22-24, 1980; Soc Am For, Washington, DC; p 214-219.

Ojima, D S; Schimel, D S; Parton, W J. 1985. Fire and ecosystem processes in the tallgrass prairie (abst only). Bull Ecol Soc Am 66(2):241.

Pearse, A S. 1943. Effects of burning-over and raking-off litter on certain soil animals in the Duke Forest. Am Midl Nat, p 406-424.

Pehl, C E. 1984. Residual effects of site preparation on growth and surface soil properties of *Pinus taeda* plantations. For Ecol & Mgt 9:1-11.

Pehl, C E; Red, J T; Shelnutt, H E. 1986. Controlled burning and land treatment influences on chemical properties of a forest soil. For Ecol & Mgt 17:119-128.

Phillips, M J; Goh, K M. 1985. Extractable soil nitrogen following clear-cutting and burning in a beech forest. Soil Sci Soc Am J 49(6):1563-1568.

Powers, R F. 1976. Principles and concepts of forest soil fertility. In 1st Ann Earth Sci Sym, USDA For Serv, CA Region, Fresno, CA.

Powers, R F. 1980. Mineralizable soil nitrogen as an index of nitrogen availability to forest trees. Soil Sci Soc Am J 44:1314-1320.

Pritchett, W L. 1976. Considerations in use of fire by prescription for managing soil and water. In Proc Fire by Presc Sym, Oct 1976, Atlanta, GA, p 33-35.

Pritchett, W L; Wells, C G. 1978. Harvesting and site preparation increase nutrient mobilization. In (Tibbin, T, ed) Proc Sym on Prin of Main Prod on Prep Sites, MS State Univ, p 98-110.

Raison, R J; Woods, P V; Jakobsen, B F; Bary, G A V. 1986. Soil temperatures during and following low-intensity prescribed burning in a Eucalyptus pauciflora forest. Aus J Soil Res 24(1):33-47.

Ralston, C W; Hatchell, G E. 1971. Effects of prescribed burning on physical properties of soil. In Proc Presc Burning Sym, Charleston, SC, April 1971, p 68-84 [Avail from USDA For Serv, SE For Exp Stn, Asheville, NC].

Richeson. 1981. Nitrogen scenarios. USDI Bur Land Mgt, Eugene, OR.

Richter, D D; Ralston, C W; Harms, W R. 1982. Prescribed fire: effects on water quality and forest nutrient cycling. Science 215:661-663.

Roehl, J W. 1962. Sediment source areas, delivery ratios, and influencing morphological factors. IASH Comm of Land Eros, Pub 59:202-213.

Romancier, R M. 1960. Reduction of fuel accumulations with fire. USDA For Serv Res Note SE-143, 2 p.

Ryan, M G; Covington, W W. 1986. Effect of a prescribed burn in ponderosa pine on inorganic nitrogen concentrations of mineral soil. USDA For Serv Res Note RM-464, 6 p.

Sampson, A W. 1944. Effect of chaparral burning on soil erosion and soil moisture relations. Ecology 25(2):171-191.

Sanders, B M; Van Lear, D H. 1988. Pre- and post-burn photo series for pine-hardwood logging slash in the southern Appalachians. 9th Conf Fire & For Meteorol, Apr 21-24, 1987, San Diego, CA:41-48.

Sandberg, D V. 1980. Duff reduction by prescribed underburning in Douglas-fir. USDA For Serv Res Pap PNW-272.

Schoch, P; Binkley, D. 1986. Prescribed burning increased nitrogen availability in a mature loblolly pine stand. For Ecol & Mgt 14:13-22.

Scholl, D G. 1975. Soil wettability and fire in Arizona chaparral. Soil Sci Soc Am Proc 39(2):356-361.

Schumm, S A. 1977. The fluvial system. John Wiley & Sons, NY, p 70-74.

Scott, V H; Burgy, R H. 1956. Effects of heat and brush burning on the physical properties of certain upland soils that influence infiltration. Soil Sci 82:63-70.

Settergren, C. D.; Krstansky, J. J. 1987. Hydrograph responses to timber harvesting in the Missouri Ozarks. In: Hay, R. L.; Woods, F. W.; De Selm, H., eds. Proceedings of the central hardwood forest conference VI; February 24-26, 1987; Knoxville, TN. Knoxville, TN: Univ Tenn & Tenn Dept For, Wildlife & Fisheries; 503-506.

Smith, P W. 1970. Concentrations of soil nutrients before and after fire. Can Jour Soil Sci 50(1):17-29.

Stednick, J D; Tripp, L N; McDonald, R J. 1982. Slash burning effects on soil and water chemistry in southeastern Alaska. Jour Soil & Water Cons 37(2):126-128.

Stransky, J J; Halls, L K; Watterson, K G. 1983. Soil response to clearcutting and site preparation. In Proc 2nd Bien S Silv Res Conf (E.P. Stone, ed), Nov 4-5 1982, Atlanta, GA; USDA For Serv Gen Tech Rep SE-24, p 54-58.

Suman, R F; Carter, R L. 1954. Burning and grazing have little effect on chemical properties of Coastal Plain forest soils. USDA For Serv, SE For Exp Stn, Res Note 56, 2 p.

Swank, W T; Waide, J B. 1980. Interpretation of nutrient cycling research in a management context: evaluating potential effects of alternative management strategies on site productivity. In Forests: Fresh Perspectives from Ecosys Anal, Proc 40th Ann Biol Colluq, OR State Univ, p 137-157.

Swift, L W. 1986. Filter strip widths for forest roads in the southern Appalachians. South Jour Appl For 10:27-34.

Switzer, G L; Nelson, L E; Smith, W H. 1968. The mineral cycle in forest stands. In For Fertil--Theory and Practice, TVA, Muscle Shoals, AL; p 1-9.

Tarrant, R F. 1956a. Effects of slash burning on some physical soil properties. For Sci 2(1):18-22.

Tarrant, R F. 1956b. Effects of slash burning on some soils of the Douglas-fir region. Soil Sci Soc Am Proc 20:408-411.

Tew, D T; Morris, L A; Allen, H L; Wells, C G. 1986. Estimates of nutrient removal, displacement and loss resulting from harvest and site preparation of a Pinus taeda plantation in the Piedmont of North Carolina. For Ecol & Mgt 15(4):257-267.

Thor, E; Nichols, G M. 1974. Some effects of fire on litter, soil, and hardwood regeneration. Proc Tall Timbers Conf 13:317-329.

Tiedemann, A R; Conrad, C E; Dieterich, J H; Hornbeck, J W; Megahan, W F; Viereck, L A; Wade, D D. 1979. Effects of fire on water: a state-of-knowledge review. USDA For Serv Gen Tech Rep WO-10, 28 p.

Tothill, J C; Shaw, N H. 1968. Temperatures under fires in bunch spear grass pastures of Southeast Queensland. J Aust Inst Agr Sci 34:94-97.

Ursic, S J. 1969. Hydrologic effects of prescribed burning on abandoned fields in northern Mississippi. USDA For Serv Res Pap SO-46, 20 p.

Ursic, S J. 1970. Hydrologic effects of prescribed burning and deadening upland hardwoods in northern Mississippi. USDA For Serv Res Pap SO-54, 15 p.

Vance, E D; Henderson, G S. 1984. Soil nitrogen availability following long-term burning in an oak-hickory forest. Soil Sci Soc Am Jour 48:184-190.

Vance, E D; Henderson, G S; Blevins, D G. 1983. Nonsymbiotic nitrogen fixation in an oak-hickory forest following long-term prescribed burning. Soil Sci 47(1):134-137.

Van Lear, D H; Danielovich, S J. 1988. Soil movement after broadcast burning in the southern Appalachians. South Jour Appl For 12:49-53.

Van Lear D H; Douglass, J E; Cox, S K; Augspurger, M K. 1985. Sediment and nutrient export in runoff from burned and harvested pine watersheds in the South Carolina Piedmont. Jour Env Qual 14(2):169-174.

Van Lear, D H; Swank, W T; Douglass, J E; Waide, J B. 1983. Forest management practices and the nutrient status of a loblolly pine plantation. IUFRO Sym on For Site and Cont Prod, USDA For Serv Gen Tech Rep PNW-163:252-258.

Van Lear, D H; Waldrop, T A. 1987. Current practices and recent advances in prescribed burning. In (Carpenter, S B, ed) Proc South For Sym, Nov 19-21, Atlanta, GA. Nat Assoc Prof For Schools & Colleges, South Reg, OK State Univ, Stillwater OK.

Van Wagner, C E. 1970. Temperature gradients in duff and soil during prescribed fires. Bi-Monthly Res Notes 26(4):42.

Vitousek, P M; Matson, P A. 1985a. Intensive harvesting and site preparation decrease soil nitrogen availability in young plantations. South Jour Appl For 9(2):120-125.

Vitousek, P M; Matson, P A. 1985b. Disturbance, nitrogen availability, and nitrogen losses in an intensively managed loblolly pine plantation. Ecology 66(4):1360-1376.

Wahlenberg, W G. 1935. Effect of fire and grazing on soil properties and the natural reproduction of longleaf pine. J For 33(3):331-337.

Wahlenberg, W G; Greens, S W; Reed, H R. 1939. Effect of fire and cattle grazing on longleaf pine lands as studied at McNeill, MS. USDA Tech Bull 683, 52 p.

Waldrop, T A; Van Lear, D H; Lloyd, F T; Harms, W R. 1987. Long-term studies of prescribed burning in loblolly pine forests of the southeastern Coastal Plain. USDA For Serv, Gen Tech Rep SE-45, 23 p.

Wells, C G. 1971. Effects of prescribed burning on soil chemical properties and nutrient availability. In Proc Presc Burning Sym, Charleston, SC, April 1971, p 86-97 [Avail from USDA For Serv, SE For Exp Stn, Asheville, NC].

Wells, C G; Campbell, R E; DeBano, L F; Lewis, C E; Fredriksen, R L; Franklin, E C; Froelich, R C; Dunn, P H. 1979. Effects of fire on soil: a state-of-knowledge review. USDA For Serv Gen Tech Rep WO-7, 34 p.

Wells, C G; Morris, L A. 1982. Maintenance and improvement of soil productivity. In Proc Sym Loblolly Pine Ecos (E Reg), Raleigh, NC, Dec 1982, p 306-318.

Wells, W G. 1981. Some effects of brushfires on erosion processes in coastal Southern California. In Eros and Sed Transp in Pac Rim Steeplands, IAHS Pub 132.

White, E M; Thompson, W W; Gartner, F R. 1973. Heat effects on nutrient release from soils under ponderosa pine. Jour Range Mgt 26(1):22-24.

Wilbur, R B; Christensen, N L. 1983. Effects of fire on nutrient availability in a North Carolina Coastal Plain pocosin. Amer Midland Nat 110(1):54-60.

Youngberg, C T. 1953. Slash burning and soil organic matter maintenance. Jour For 51:202-203.

Youngberg, C T. 1982. Soil-site relationships. Mapleton Ranger Dist, Siuslaw NF.

Youngberg, C T; Wollum, A G. 1976. Nitrogen accretion in developing Ceanothus velutinus stands. Soil Sci Soc Amer Jour 40:109-112.

Effects of Herbicides on Soil Productivity and Water Quality

APPENDIX C

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EFFECT OF HERBICIDES ON SOIL PRODUCTIVITY AND WATER QUALITY

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INTRODUCTION

The southern yellow pine and hardwood forests of the South constitute some of the most intensively managed forest ecosystems in the world (Stone 1983; Kellison and Gingrich 1982). These forests also occur in a region with one of the fastest growing human populations in the United States. Furthermore, future resource demands in the South will certainly intensify as the population expands and the forest land base shrinks. The whole mix of public and private forest resources including wood, wildlife, recreation, range, and water will need intensive management to meet increased demands. One crucial concern resulting from this intensification of forest management is the potential effect of silvicultural practices on water and soil resources.

Intensive forest vegetation management practices such as short rotations, clearcut harvesting, mechanical site preparation, burning, drainage, and fertilization created concerns in the early 1970's about possible adverse impacts to soil and water resources. Research during the past two decades has demonstrated the range of environmental effects of these practices. If properly prescribed, applied, and guided, these practices can be conducted while conserving valuable soil and water resources. In addition, over the past 5 to 10 years a further intensification of vegetation management has involved increased use of herbicides. These chemicals are now used extensively across the South's managed forests to control weed competition, reduce the use of soil-disturbing mechanical site preparation techniques, and increase tree growth. The driving force behind increased herbicide use has been the need to improve growth on a diminishing forest land base at a lower initial investment cost. In addition, herbicides are being used to minimize the soil displacement and erosion losses that adversely affect site productivity and water quality. Ten years ago, herbicide use was very scattered and mainly in a testing mode. Now it is a widespread practice. Nearly all public and private forestry organizations have operational programs for suitable stands.

The use of herbicides in the South is not new since agriculture has a long history of pesticide applications. However, increased herbicide use in forest management has occurred precisely when states within the region have recognized potential and actual water pollution risks from agricultural pesticides. Thus, the general public and resource managers have questioned the use of herbicides for vegetation management on forests which are sources for much of the South's streamflow and ground water.

The purpose of this paper is to review the effects of herbicides on soil productivity and water quality. This is accomplished by discussion of herbicide characteristics, applications, and environmental interactions as they influence effects on soil productivity and surface and ground water quality. Soil productivity effects are discussed in a general context. In regard to water quality, specific information and research results from Southern studies are used, where possible, for the individual herbicides considered in this EIS.

HERBICIDE USE

Forestry herbicides can affect non-target plants and animals, and surface and ground water quality at several stages in the use cycle. These stages consist of (1) storage, (2) transportation, (3) loading and mixing, (4) application, (5) equipment cleanup, and (6) container disposal. During and after application, herbicide residues usually move onto the landscape in a diffused nonpoint source pattern. It is during this phase that most public concerns for non-target organisms and water quality arise. The other 5 stages of herbicide use usually deal with concentrates which constitute potential point sources of environmental pollution. These stages have historically caused the most environmental problems. A number of publications are available which discuss safe handling of herbicides during all phases of chemical use (Neary and Taylor 1984; Singer 1980; USDA-USEPA 1975).

Most environmental fate and impact studies conducted on forestry herbicides have focused on off-site movement during and after application. It is during this stage of herbicide use that most adverse public reactions and concerns for environmental quality occur. The bulk of this paper will deal precisely with this aspect. However, references will be made to problems with concentrated materials.

ECOSYSTEM FATE

When herbicides are applied to forest ecosystems, a number of processes affect the environmental fate and impact of these chemicals. Understanding these processes is important to determining the environmental impact of herbicide use in vegetation management programs. To reach such an understanding, we must consider the important zones and processes involved in herbicide application, movement and transformation (figure 1). The key environmental zones are the atmosphere, above-ground vegetation, soil surface, soil rooting zone, unsaturated zone below the rooting depth, and ground water.

Herbicides and their breakdown products are transported within ecosystems mainly through the water cycle. Precipitation, evaporation, runoff, leaching, and root uptake are the major water pathways. Within the unsaturated and saturated soil zones and geologic strata, movement can be lateral, upward, or downward. These processes, as they operate in forested watersheds, are discussed in great detail by Hewlett (1982), Anderson and others (1976), and Crossley and Swank (1987). Runoff, leaching, root uptake, and movement in soil and ground water are the primary hydrologic processes governing herbicide movement. Precipitation and evaporation are the principal driving forces in the water cycle.

A variety of processes occur within the environmental zones which affect the gain or loss of herbicide residues within the system (figure 1). The importance of these processes on any given site is determined by individual herbicide characteristics, climatic factors, soil-water properties, and indigenous organisms. These processes have been analyzed and discussed in considerable detail (Hance 1980; Grover 1988). The purpose of the discussion here is to give the reader an overview of these key environmental fate processes.

HERBICIDE CHARACTERISTICS

The important characteristics which distinguish herbicides and their potential effects on the environment are listed in table 1. Formulation, solubility, and vapor pressure are the key physical characteristics of herbicides which affect environmental fate. The other characteristics listed in this table involve interactions with the environment and are discussed later.

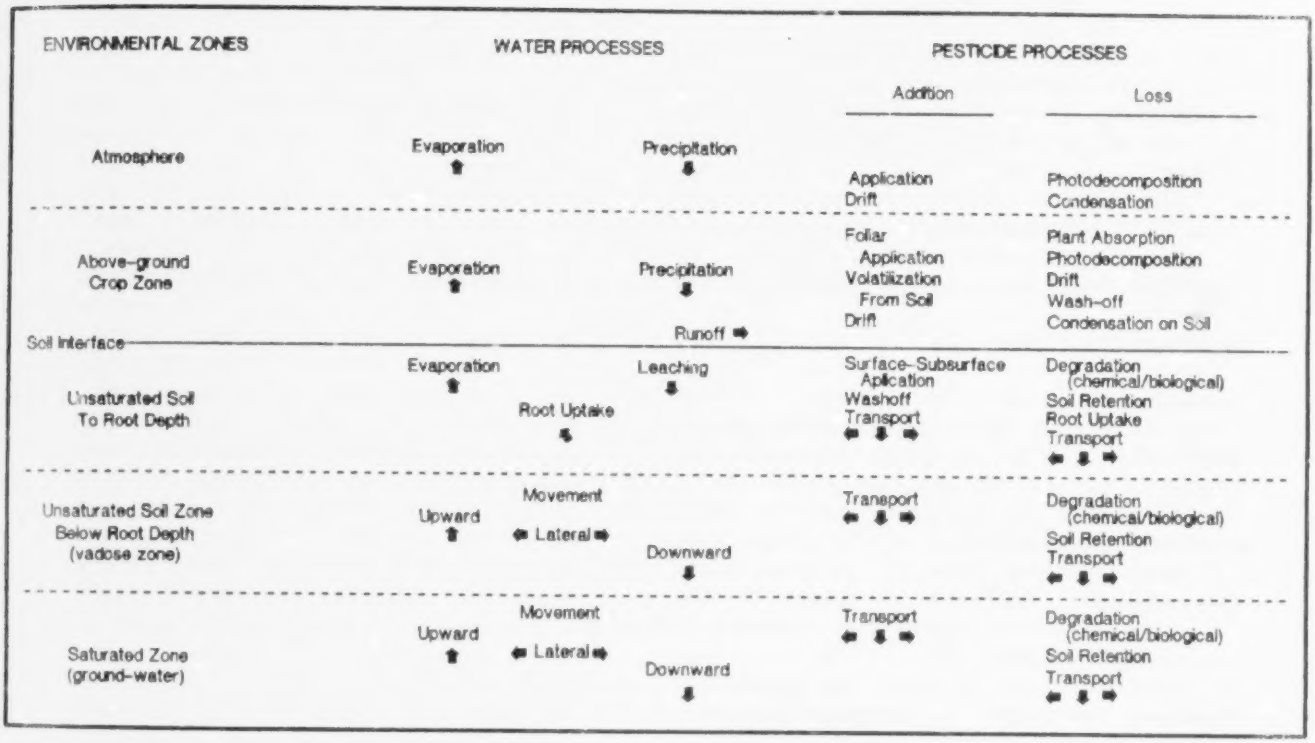
ENVIRONMENTAL ZONES
WATER PROCESSES
PESTICIDE PROCESSES
Addition
Loss
Atmosphere
Evaporation ↑
Precipitation ↓
Application
Drift
Photodecomposition
Condensation
Above-ground Crop Zone
Evaporation ↑
Precipitation ↓
Foliar Application
Volatilization From Soil
Drift
Plant Absorption
Photodecomposition
Drift
Wash-off
Condensation on Soil
Soil Interface
Runoff →
Unsaturated Soil To Root Depth
Evaporation ↑
Leaching ↓
Root Uptake
Surface-Subsurface Aplication
Washoff
Transport ← ↓ →
Degradation (chemical/biological)
Soil Retention
Root Uptake
Transport ← ↓ →
Unsaturated Soil Zone Below Root Depth (vadose zone)
Movement
Upward ↑
← Lateral →
Downward ↓
Transport ← ↓ →
Degradation (chemical/biological)
Soil Retention
Transport ← ↓ →
Saturated Zone (ground-water)
Movement
Upward ↑
← Lateral →
Downward ↓
Transport ← ↓ →
Degradation (chemical/biological)
Soil Retention
Transport ← ↓ →

Figure 1.--Processes affecting water movement and pesticide transport in various environmental zones.

The formulation of a herbicide consists of the active ingredient and inert carrier materials. Chemical manufacturers mix these materials into their trade formulation to provide easy application and efficient weed control. Variations in formulations can be due to changes in either the active ingredient or the inert materials. The whole range of formulations have been discussed in detail (Sassman and others 1984). The inert carrier materials are "inert" only with regard to their herbicidal properties. They range from clay to petroleum solvents. Like all chemicals, their effects on plants and animals can vary. Formulations are important since changes by individual chemical manufacturers can affect the other two physical characteristics, solubility and vapor pressure. The most commonly used forestry herbicide formulations are liquid concentrates, wettable powders, granules, pellets, emulsifiable concentrates, and soluble powders (Neary 1985b). The type of formulation for a particular herbicide also affects the application system and the potential for off-site movement.

Table 1.--Important environmental characteristics of the silvicultural herbicides in Region 8

Name	Solubility 25 C	Half-Life[1]	Photo-Degradation	Microbial Degradation	Hydro-lysis	Volatil.	Adsorp. Coeff.	LD50[6]	LD50[7]
	ppm	days						mg/kg	mg/kg
2,4-D	3,000,000[2]	28	Minor	Yes	Yes	Yes (F)[8]	0.5[9]	375	168 (F)
2,4-DP	710	10	Minor	Yes	No	Yes (F)	0.5	532	1
Dicamba	4,500	25	No	Yes	No	No	0.1	757	135
Fosamine	1,790,000	<10	No	Yes	No	No	20.0	24,400	670
Glyphosate	12,000	61	Minor	Yes	No	Low	16.5	4,320	1,000
Hexazinone	33,000	30	Yes	Yes	No	Low	0.2	1,690	370
Imazapyr	15,000	30	Yes	Yes	No	No	0.3	5,000	100
LFO[10]	---[3]	6	No	Yes	No	Yes	0.8	28,000[11] 7,380	1
Picloram	430,000[4]	63	Yes	Yes	Yes	No	0.6	8,200	21
Sulfometuron	300[5]	10	No	Yes	Yes	No	0.7	5,000	12
Tebuthiuron	2,500	392	No	Yes	No	Low	2.4	644	112
Triclopyr	430	46	Rapid	Yes	No	Low	1.5	530	148

1 Average half-life
2 Amine salt formulation
3 Light Fuel Oil is not water soluble
4 Water solubility for potassium salt
5 Solubility at pH 7; 10 ppm at pH 5
6 LD50, technical grade, for rats; for formulation
7 LC50 for bluegill sunfish, 96 hrs, see appendix A, tables 6-8 to 6-19
8 Formulated product
9 See appendix A, chapter 4
10 Light fuel oil
11 First figure kerosene and the second diesel

Herbicide formulation can directly affect solubility. An example is 2,4-D; the dimethylamine salt of 2,4-D is totally water soluble while the butoxyethanol ester of 2,4-D is essentially insoluble in water (WSSA 1986). The solubility of herbicide active ingredients in water is also one index of potential for off-site movement. In general, herbicides with high water solubility have the greatest potential to move by storm runoff into streams and lakes or by deep leaching into an aquifer. Some exceptions occur when herbicides interact strongly with the soil chemical/biological system. All of the herbicides discussed in this EIS are fairly soluble (table 1), and some are very soluble (fosamine, hexazinone, imazapyr, picloram, and tebuthiuron). Glyphosate is an exception to the solubility - transport rule-of-thumb. Although it is readily soluble in water, its potential to move is very low since it is strongly adsorbed onto organic matter in the soil.

Most of the forestry herbicides have low vapor pressures and thus are not prone to volatilization losses (table 1). In addition, many are in stable solid formulations (i.e. pellets, granules, soluble powders, and wettable powders). Herbicides in liquid formulations are mainly non-volatile salts or low-volatile esters.

APPLICATION

Application systems for forestry herbicides are discussed in some detail in the Risk Assessment (appendix A, chapter II) and elsewhere (Cantrell 1985; Miller and Williamson 1987). The environmental effects of herbicides are influenced strongly by application conditions including placement, system, formulation, rate, timing, use pattern, and buffers. Other things being equal, it is mainly the prescription, application, and execution which determine the severity of environmental impacts. There are almost infinite combinations of these factors to consider. Our purpose is to briefly discuss some of the important concepts and comparisons.

PLACEMENT: Herbicides can be placed on the foliage or stems of target plants, on the soil, or directly into stems. Foliar application generally involves a greater hazard because herbicides are spread through the air. They can be moved around by aerial drift, washed off plant leaf surfaces, or physically dislodged. Soil applications may result in a lower hazard of off-site movement, but introduce additional problems of runoff and leaching. Soil-active herbicides usually do not enter the target plants as rapidly as foliar ones. Drift potential is reduced to near zero if solid formulations are used. The least potential hazard comes from direct injection into the target plant since nearly all the herbicide is placed where washoff, runoff, and drift does not occur. However, careless cleanup of equipment may result in water contamination by direct runoff into streams or leaching into wells.

SYSTEMS: Various herbicide-application systems are commercially available (Cantrell 1985). The choice of system affects the potential environmental impact and fate of a herbicide. For instance, ground spray systems are not subject to the same drift problems as aerial ones although drift can still occur. Among aerial application methods, helicopters give a greater degree of placement control than fixed-wing aircraft. The type of nozzles selected for spray equipment and the operating pressure of the system directly affect droplet size, distribution pattern and drift potential. A more complete discussion of this topic is found in chapter II, section D of the EIS.

FORMULATIONS: The chemical formulation of a herbicide can also affect environmental fate. For instance, hexazinone is available in a solid as well as a liquid formulation. For aerial application, the solid formulation is much easier to control. Except for short-distance dust movement, drift is not a hazard with aerial application of solid formulations. Also, differences in the chemical properties of different liquid formulations can affect environmental fate and movement.

RATE: Herbicide application rate strongly affects environmental impact and fate. Rates can vary from 0.1 to 5.0 kg/ha (1 oz/ac to 5 lb/ac) active ingredient, depending on the herbicide and target vegetation. Obviously, with a low rate of herbicide application, residues will dissipate faster, potential exposure of non-target organisms will be lower, and the amount of chemical available for off-site transport into surface water or ground water will be less. Selecting herbicides which will effectively control target weeds at low application rates reduces potential adverse environmental impact.

TIMING: Timing of herbicide applications in relation to climatic conditions and the growing condition of vegetation is important. Often timing is the difference between safe and unsafe use of the same herbicide. This difference can be a matter of seasonal, daily, or hourly timing. Application of a highly soluble herbicide during a dry period with few and low intensity storms presents a far different hazard to water quality than during a rainy season. The same contrast occurs between clear versus rainy or foggy days. Herbicide applications during early morning hours with light winds, or mid-day when winds are gusty, present two different hazard levels.

USE PATTERN: Another important factor determining the environmental impact of herbicides is their use pattern. Generally, forestry use of herbicides is of low intensity compared with agricultural use. Forestry herbicides are normally applied once or twice in a 25- to 75-year rotation. Agricultural usage is yearly or even monthly during the growing season. The current level of herbicide use on national forests in Region 8 (appendix A, chapter 1) involves annual applications to only 1 percent of the national forest land base. The two types of special use areas that have herbicide use patterns similar to agriculture are seed orchards and nurseries. They occupy very small land areas and are being handled with separate environmental impact statements.

BUFFERS: The presence and size of buffers has a large effect on the potential impact of herbicides on water quality. Buffers are used as a mitigation measure to reduce or prevent herbicide movement into water. The size of buffer needed is a function of the chemistry of each herbicide , the application system, and the sensitivity of the water resource. Effect of buffer size is discussed in later sections and in the Risk Assessment (appendix A).

DISAPPEARANCE OF HERBICIDE RESIDUES

Once a herbicide is applied to a site, it is subjected to natural processes eventually resulting in its disappearance. The herbicides initially are retained on-site by being deposited on foliage and litter surfaces, placed directly into vegetation, applied within an inert granule carrier, or adsorbed onto soil surfaces. Their disappearance is a combination of two groups of processes, transport and degradation.

RETENTION PROCESSES: These processes are important in assuring either that the herbicide gets to its target or is kept on the treated site. Foliar penetration is a key process in getting herbicides through the waxy leaf surface and into the target plant. There are many kinds of adjuvants or herbicide formulations which aid this process. Injecting herbicides directly into trees is one obvious way of easily getting herbicides to the target. Herbicides applied in granular formulations are easily placed on-site and then held there until rainfall can disperse the active ingredient. Once herbicides enter the soil, adsorption is an important process. Organic matter content is very important in determining adsorption as it relates directly to the soil's ability to retain chemical residues. The higher the organic matter content of the soil the greater the potential to retain herbicide residues.

TRANSPORT PROCESSES: Herbicide transport processes include drift, foliar and stem washoff (also physical dislodgment), volatilization, plant uptake, leaching, surface runoff, and subsurface flow. Through these processes, herbicides move within a treated area and from target vegetation to water or non-target organisms. All these movement processes are affected by a complex set of chemical, physical, climatic, hydrologic, edaphic, and biologic factors.

Drift is the movement of herbicides in air as suspended droplets or dust. Rainfall can cause foliar and stem washoff after herbicide application, removing herbicide residues from plant surfaces and transporting them to the soil. Volatilization occurs while herbicides are still exposed to sunlight and air, and involves chemical movement in the vapor phase in air. Plant uptake removes herbicides from foliage and bark surfaces or from the soil, and temporarily or permanently, depending on the herbicide, removes them from transport. Leaching moves herbicides through litter, soil, and out of the plant rooting zone. Surface runoff rapidly transports residues off-site either in solution or adsorbed to sediment. Subsurface flow of water removes herbicides in solution from the treatment site in slower ground water flow.

DEGRADATION PROCESSES: Processes that break down herbicide chemical structures include photodecomposition, microbial and plant metabolism, thermal degradation, and hydrolysis. These processes, along with those that transport herbicides, determine the degree to which a herbicide persists in the environment. Herbicide persistence is advantageous for controlling target vegetation, but can be a disadvantage because of movement off-site or toxicity to subsequently planted trees.

Some herbicides readily photodegrade, some do not, and some do so only in water. There are many micro-organisms in the soil that can utilize herbicides as energy sources and break down these chemicals into simpler structures. In addition, plants can alter herbicide structures while the herbicides are affecting the plant's physiology. Herbicides are also degraded into simpler compounds by physical-chemical processes like hydrolysis.

SOIL PRODUCTIVITY

ISSUE: One concern about herbicide use in southern forests is their long-term effect on soil productivity. Does introduction of synthetic chemicals into the forest vegetation and soil system produce adverse, advantageous, or neutral changes? This question can be answered, in part, by examining tree growth responses, erosion effects, and soil micro-organism impacts.

TREE GROWTH

Many studies clearly demonstrate that tree growth responds positively to herbicide applications in the South (Bacon and Zedaker 1987; Knowe and others 1985; Nelson and others 1981; Swindel and others In Press). Application mistakes can cause tree mortality, but the vast majority of experience is with successful treatment results. Elimination of competing plants early in a stand's rotation can have significant impact on short-term and long-term productivity (Glover 1985; Michael 1980, 1985a; Swindel and others In Press). Plant nutrients are in short supply in many soil types of the South due to past land use abuses or pedogenic factors. The body of information available now indicates that herbicide use can significantly increase forest productivity (Neary and others In Press).

EROSION

Excessive erosion is currently degrading the productivity of many agricultural soils in the South (Larsen and others 1983). Many forest stands in this region were established on sites that were eroded and impoverished by abusive agricultural practices. Our present forests stabilized eroding soils and have been rebuilding productivity over the past 50 - 80 years.

Erosion and soil dislocations within sites have been identified as potential negative impacts on future forest productivity in the South (Neary and others 1984). The litter and surface soil horizons are crucial for the maintenance of site productivity. The bulk of the nutrients that promote good tree growth are found in these surface layers. Any activities which remove or redistribute these horizons can be potentially damaging to forest productivity. Mechanical site preparation (Beasley 1979; Douglass and Goodwin 1980) and burning (Douglass and Van Lear 1980) have been traditionally practiced to remove obstructions, eliminate competition, and prepare sites for planting. However, intensive mechanical site preparation has been identified as a major factor adversely affecting site productivity.

Herbicide use for site preparation, even in steeper terrain, causes very little erosion and maintains good hydrologic conditions. Herbicides do not disturb the soil and usually leave a good litter layer which mitigates raindrop impact, promotes infiltration, and greatly reduces erosion. Examining erosion from a variety of site preparation techniques and locations in the South, it is evident that herbicide use results in sediment yields more similar to undisturbed watersheds than mechanically prepared ones (table 2).

Evidence on erosion clearly points to the benefits of herbicide use in southern forestry. This is true first from the viewpoint of reducing adverse site productivity by maintaining scarce nutrients. It is also true regarding water quality impacts. Sediment is the biggest water quality problem in the region (Larsen and others 1983) and herbicides show a very positive effect (Neary and others 1986).

Table 2.--Sediment yields from forest watersheds in the South during the first year after site preparation

Reference	Treatment	Sediment Loss Mass	 % of Control	Physio-graphic Province[1]
		kg/ha		
Neary and others 1986	Control	67	---	P
	Herbicide	170	254	
Douglass and Van Lear 1983	Control	39	---	P
	Burned	44	113	
Douglass and Goodwin 1980	Control	35	---	P
	Kg,Disk,Grass	720	2,057	
	Kg	3,501	10,000	
	Kg,Disk	9,730	28,700	
Beasley 1979	Control	620	---	UCP
	Chop	12,540	2,023	
	Shear	12,800	2,065	
	Bed	14,250	2,298	
Beasley and others 1986	Control	147[2]	---	UCP
	Shear, Wind.	1,005	684	
	Herbicide	205	139	
Riekerk, 1982; and Neary and others 1982	Control	3	---	LCP
	Burn, Bed	7	233	
	Windrow & Bed	36	1,200	

[1] P = Piedmont; UCP = Upper Coastal Plain; and LCP = Lower Coastal Plain

[2] Second year data used due to very high stormflow

SOIL ORGANISMS

Does herbicide use adversely affect soil flora and fauna? Certainly the removal of a live vegetation canopy has significant effects on the thermal and moisture regimes of the forest floor and soil horizons. But the resulting changes in soil organisms are due more to physical than chemical effects (Mayack and others 1982).

The micro- and macro-organisms found in the forest floor and soil horizons play very important roles in the functioning of forest ecosystems. They are important in processes such as organic matter decomposition, nutrient mineralization, nitrogen transformations, respiration, soil structure and porosity formation, etc. Overviews of herbicide effects on soil organisms are provided by Eijasackers and van de Bund (1980), Greaves and Malkoney (1980), Greaves and others (1976), and Martin (1963). Although stimulatory as well as inhibitory responses have been observed in micro-organisms, much remains to be learned about the complex interactions between soil organisms and herbicides. Effects are very much dependent on the herbicide, application rate, and soil environment factors. Where adverse effects have been observed, herbicide concentrations exceeded those measured under actual operational conditions (Fletcher and Friedman 1986). There is, however, a general consensus that herbicide usage at normal forestry rates does not reduce the activity of micro-organisms.

CONCLUSIONS: There is no evidence that the herbicides currently used in forest management in the South produce any adverse effects on site and soil productivity. There is substantial evidence that herbicide usage as a silvicultural tool can increase site productivity.

WATER QUALITY

The occurrence and significance of herbicide residues in surface waters result from a complex set of factors. Occurrence depends on the type and location of surface water, mixing and dilution of streamflow, herbicide properties such as solubility and degradation potential, method and timing of application, timing and amount of rainfall, site characteristics, and soil properties. The biological significance of a residue concentration depends upon water usage, toxicity levels, and exposure. The legal significance depends upon water quality standards.

OCCURRENCE

The concentrations of herbicides in surface waters depend largely on the type of water and location in relation to the application area. Streams generally have the most variable concentrations, and surface flow from first-order drainages contains the highest residue concentrations. Streams receiving herbicide residues in flow from ephemeral channels generally have concentrations one to two orders of magnitude higher than those receiving only subsurface flow. Wetlands close to treatment areas may contain higher residue levels because of their small size and lack of flushing. Herbicide concentrations in lakes depend on residue inputs, lake size, and recharge by ground water or streamflow.

Mixing and dilution are very important in determining amount and duration of herbicide residues in surface waters. Neary and others (1983) measured hexazinone concentrations that averaged 0.442 ppm (mg/L) in stormflow from 2.5 ac (1 ha) ephemeral watersheds, but were less than 0.002 ppm during the same storm downstream at a 250 ac (100 ha) watershed. This resulted in an actual dilution factor of 221 compared to a straight area ratio of 100. Within large watersheds (50,000 ac or about 20,000 ha) entirely under intensive silviculture, dilution factors for forestry herbicide residues could range from 30 to 45,000 times. The former value is a straight area ratio based on herbicide treatment of each unit area of land once in a 30 year rotation. The latter value is based on only one unit area (1 ha or 2.5 ac) of the large watershed being treated with one particular herbicide and application of the field-measured dilution factor (221).

Herbicide properties such as use rate, solubility, adsorption coefficient, and half-life are very significant in determining the amounts of residues which enter into surface waters. Herbicides with a typical use rate of greater than 4.0 kg/ha (greater than 3.6 lb/ac) are more likely to be detected in surface flow than those used at less than 0.40 kg/ha (less than 0.36 lb/ac). Solubility is a general index of potential to move in water, but there are exceptions. Positively charged glyphosate is highly soluble in water but generally does not move off-site to any appreciable extent since it is quickly adsorbed to organic matter in the soil and immobilized. Negatively charged picloram is highly soluble and easily mobile. Although picloram can be adsorbed to the soil it is readily desorbed and mobilized. A herbicide like sulfometuron methyl, with a short half-life of less than 10 days, is less likely to move into surface water than tebuthiuron (half-life of 392 days)(table 1). Herbicides subject to photodegradation are also less likely to be found in surface water.

The method and timing of applications is extremely important. Generally, the risk of water pollution is less with ground applications than aerial ones, and granular formulations are easier to control than liquid formulations. The type of equipment used and the timing in relation to climatic and vegetation variables are also critical.

Rainfall timing, amount, and intensity affect herbicide concentrations in streamflow. These effects are very much a result of the type of hydrologic response (surface runoff versus subsurface flow). Very large storms (greater than 25 year return period) generally do not result in high herbicide concentrations because of dilution by large flow volumes. Likewise, small storms (less than 1 month return period) may not produce sufficient stormflow. It is the intermediate storms that produce the higher concentrations.

Site characteristics like topography, treatment-area size in relation to watershed area, and distance to nearest perennial stream are other factors affecting occurrence of herbicide residues in surface waters. Soil characteristics are also important. Organic matter is the most important factor. Soils high in organic matter have a large potential to retain herbicide residues in an adsorbed condition while soils low in organic matter like sands have a low capacity to hold herbicide residues within the soil profile.

SIGNIFICANCE

If herbicide residues enter surface or ground water, their significance is determined by residue duration, water usage, chemical toxicity, and potential exposure of humans, animals, or plants. For many herbicides there are no water quality standards because of their low toxicity, the infrequency of their occurrence in drinking water supplies, and the recent nature of their use in forests. Herbicides such as 2,4-D (0.100 ppm) and picloram (1.050 ppm) have established drinking water quality standards (NRC 1983).

One important issue to consider is the distinction between contamination and pollution. All water is contaminated. That is, no surface or ground water is pure. All water contains varying levels of other elements or compounds. On the other hand, water is normally considered polluted only when concentrations of contaminants exceed a water quality standard and threaten some use of the water. In the case of herbicide residues in water, the scientific contamination/pollution distinction often conflicts with individual perceptions of risk. As analytical instrumentation and techniques improve, herbicide residues are being measured at lower concentrations. Detection of herbicide residues, other compounds, or elements does not imply that pollution has occurred or that a health risk exists. Thus, objective evaluations of the significance of short-duration, low-level concentrations of herbicides in water must be made.

HERBICIDE RESIDUES IN SURFACE WATERS

The remainder of this section will discuss the occurrence of herbicide residues in surface water. Data from the South will be used where they are available. References from other forest ecosystems will be used to augment these data where information on particular chemicals is lacking.

2,4-D: This is one of the phenoxy herbicides that functions as a plant growth regulator. Since its introduction into forestry in the late 1940's, it has become the most widely used and intensively studied forestry herbicide still in use (Norris 1981a). A large variety of formulations are available commercially (Sassman and others 1984). Salt formulations are readily absorbed through the roots of weeds, and ester formulations are most easily absorbed through the foliage.

Toxicological studies indicate that most formulations are mildly toxic to mammals and birds (table 1). 2,4-D does not bioaccumulate to any appreciable extent. It is highly soluble in water and is translocated and metabolized readily within plants. Persistence of 2,4-D in forest soils is rather short (less than 4 weeks) as it is degraded by microbes, translocated into plants, and photodegraded to a limited extent (Norris 1981b). Volatilization is dependent on formulation. Transport losses from soils to water are mediated by organic matter, low surface runoff in most forest soils, and moderately rapid microbiological degradation.

A review of 2,4-D residues in water after forestry applications in the Pacific Northwest indicated that 90 percent of the streamflow samples contained no 2,4-D and the remainder had an average concentration of less than 0.040 ppm (Sassman and others 1984). 2,4-D was applied to all but a narrow (less than 5 m) buffer strip of Watershed 6 (9 ha or 22 ac) at the Coweeta Hydrologic Laboratory in western North Carolina (Douglass and others 1969). Application of 3.4 kg/ha (3.0 lb/ac) in 760 L of water carrier by a ground spray system did not result in any detectable 2,4-D in the stream.

Throughout the South, 2,4-D is used for injection of hardwood stems. This application method is less hazardous than spraying and is the commonest 2,4-D application method in national forests. A recent operational monitoring of 2,4-D injections in Alabama, Georgia, Tennessee, and Kentucky did not detect residues of this herbicide in streamflow from treated watersheds. In most of these applications, minimum buffers of 9.1 m (30 ft) were maintained.

2,4-DP: This herbicide is also a phenoxyacetic acid chemical and very closely related to 2,4-D (Norris 1981a). It is available in a variety of formulations like 2,4-D but is less soluble and degrades faster (table 1). There is virtually no information on the fate of 2,4-DP residues in forest watersheds in the South.

DICAMBA: This herbicide is a benzoic acid derivative used as a pre- and post-emergence treatment on broad leaved weeds and brush resistant to phenoxy compounds. It is available in several formulations including water soluble salt and granular formulations. Dicamba is readily absorbed by leaves and roots and translocated within plants. It is an auxin-like growth regulator for plants but is only slightly toxic to aquatic and terrestrial animals (table 1). Dicamba does not bioaccumulate nor photodegrade, but is readily metabolized by plants and micro-organisms (Smith and Cullimore 1975). It has a moderate half-life in soil (table 1).

Because of its high solubility and low soil adsorption (table 1), dicamba is a fairly mobile herbicide (Norris and Montgomery 1975). Spray application of 1.12 kg/ha (1.0 lb/ac) to about 25 percent of a watershed in Oregon produced a maximum stream concentration of only 0.037 ppm which was attributed to spray drift. Concentrations of dicamba in streamflow did not persist much beyond 2 days. Other studies have only measured low (less than 0.001 ppm) and infrequent concentrations of dicamba. Micro-organisms in water are very important in dicamba dissipation in surface waters (Scifres and others 1973). As with 2,4-DP there is virtually no information on dicamba movement in forest watersheds of the South. Based on data from the Pacific Northwest, dicamba residues would not be expected to be very high nor persist long because of microbiological activity (Norris 1981b).

FOSAMINE: This herbicide is a selective chemical that is absorbed, translocated, and metabolized within plants. Fosamine does not photodegrade, but degrades rapidly in soil due to microbial activity (table 1). Its short half-life is a function of rapid micro-organism metabolism and strong adsorption in soils. Fosamine does not bioaccumulate because of the ease and speed with which it is metabolized. In water, fosamine is subject to adsorption onto sediments and rapid micro-organism attack. There is virtually nothing in the literature to indicate expected fosamine concentrations in surface waters under operational use conditions, and no data exist for southern forest watersheds.

GLYPHOSATE: This is a broad spectrum herbicide that is very effective on a number of forest weed species. The isopropylamine salt formulation is soluble in water, but glyphosate is strongly adsorbed in the soil (table 1). This herbicide is readily absorbed and translocated within plants but is not metabolized. The major degradation pathway is microbial breakdown in the soil although varying rates result in a longer half-life than some of the other herbicides (table 1). Glyphosate does not photodecompose to any extent and does not volatilize (Rueppel and others 1977). It is low in toxicity to aquatic and terrestrial organisms.

Glyphosate residues up to 5.2 ppm have been measured in runoff from agricultural fields with high transport of sediment. Residues in canals from weed control with glyphosate on ditchbanks were considerably lower (0.010 ppm) (Sacher 1978). Aerial application of glyphosate to a forested watershed resulted in low initial concentrations in streamflow (0.070 ppm). No buffer strips between the perennial stream and the herbicide-treated area were used. A peak concentration of 0.550 ppm occurred 14 days after application with a rapid decline in concentrations because of micro-organism degradation (Newton and others 1984). No data are available from applications in southern forest watersheds.

HEXAZINONE: Hexazinone is a selective triazine herbicide that controls many annuals and perennials. It is a very effective and widely used forestry herbicide because many conifers can tolerate it at rates that control competition. Granular and liquid formulations are available.

Hexazinone is practically non-toxic to aquatic and terrestrial organisms and established toxicity thresholds are not experienced in the environment (table 1). Since hexazinone is readily soluble in water, it is susceptible to off-site movement by surface runoff and leaching. It is degraded by microbial action and photodecomposition (Rhodes 1980). Hexazinone is not prone to loss by volatilization. Its half-life is generally less than 30 days, but varies between 2 weeks and 6 months, depending on soil and climatic conditions. Some phytotoxic metabolites are produced by microbial degradation but they are generally short-lived.

Hexazinone fate and transport in southern forested watersheds is better documented than any of the other herbicides. Miller and Bace (1980) reported high concentrations (up to 2.400 ppm) from direct fall of hexazinone pellets into a perennial stream. The pellets were accidentally dropped when a helicopter overflew a streamside buffer zone on one pass. Concentrations fell within 24 hours to 0.110 ppm and by 10 days were down to less than 0.010 ppm. In another aerial application in Tennessee, pellets were applied to less than 20% of a large watershed but no streams were overflown (Neary 1983). Consequently, hexazinone was never detected in streamflow during a 7 month period following application.

In a more detailed study in the upper Piedmont of Georgia, four small ephemeral watersheds (1.0 ha or 2.5 ac) were broadcast-treated with hexazinone pellets at a rate of 1.68 kg/ha (1.5 lb/ac) (Neary and others 1983). For the next year surface runoff from 26 storms was collected to determine hexazinone transport in streamflow. Residues peaked in the first storm (0.442 ppm) and declined steadily thereafter. Loss of hexazinone from the treated sites averaged 0.53 percent with two storms accounting for nearly 60 percent of the off-site transport. Subsurface movement in baseflow occurred 2 months after the hexazinone pellet application, lasted for less than 2 weeks, and produced a short-term pulse with a peak of 0.024 ppm.

Hexazinone was applied to a 11.5 ha (28 ac) watershed in Arkansas as a liquid spot application with somewhat different results (Bouchard and others 1985). The application rate for this study was slightly higher than in the Georgia study, but the ephemeral channels were not treated. As a result, hexazinone residues were never detected in surface storm runoff. Baseflow from this watershed continued to carry low levels of hexazinone (less than 0.014 ppm) for over a year. Similar concentrations (0.006 to 0.036 ppm) were measured in streamflow in another set of spot treatments in Alabama and Georgia.

IMAZAPYR: This herbicide is new to southern forestry. Imazapyr comes from the imidazolinone family of chemicals and is a very effective, broad-spectrum herbicide. It is practically non-toxic to aquatic and terrestrial organisms (table 1). Imazapyr has a low adsorption coefficient and intermediate half-life of 19-34 days. It is degraded by micro-organisms, photodecomposes, and does not bioaccumulate. Imazapyr is readily absorbed through foliar and root surfaces and easily translocates to meristem tissues.

Application of imazapyr by air to 40 to 121 ha (99 to 299 ac) watersheds in Alabama produced peak streamflow concentrations of 0.130 ppm where a streamside management zone was employed (Michael 1986). However, this concentration lasted less than 4 hours, and daily average peak stream concentrations did not exceed 0.030 ppm. In 180 days of monitoring after treatment only 4 of 184 stream samples contained quantifiable residues of imazapyr.

Imazapyr half-life was determined in treated vegetation and soil in Alabama. The half-life for vegetation under field conditions ranged from 12 to 35 days and in soil from 19 to 34 days.

LIGHT FUEL OIL: There are no data in the literature on the concentrations and movement of light fuel oil in forested watersheds of the South.

PICLORAM: This herbicide belongs to the picolinic acid family of chemicals and functions similarly to the phenoxyacetic acid herbicides in mimicking growth hormones. It is very effective on many resistant woody weeds (NRCC 1974) and is used most frequently as a salt formulation in combination with 2,4-D.

Picloram and its salts are relatively nontoxic to most non-target organisms including micro-organisms, fish, and birds (table 1). Since picloram is formulated as a potassium or isopropanolamine salt, it has a high water solubility. That combined with a relatively low adsorption coefficient makes water contamination a concern with the use of picloram. This is particularly the case since many vegetable crops are sensitive to picloram at concentrations as low as 0.010 ppm (Baur and others 1972).

Losses of picloram due to volatilization are low and photodegradation occurs only in direct sunlight. Picloram is only slowly degraded by micro-organisms which is why it has one of the longer half-lives (table 1). Half-life of picloram is climate and soil dependent and can be as short as 30 days in humid-warm climates and as long as 180 days in cold-dry ones (NRCC 1974).

Picloram concentrations in streamflow have been studied extensively in a number of ecosystems (NRCC 1974). Applications to rangelands in Texas have produced peak concentrations of up to 2.170 ppm. Usually this involves surface runoff shortly after application with no buffer strip.

Picloram was manually broadcast at a rate of 5.0 kg/ha (4.5 lb/ac) to 17% of a 30 ha (74 ac) watershed in the Appalachian Mountains (Neary and others 1985). Residues of the herbicide were measured in soil solution on the treatment site at concentrations up to 0.350 ppm. A 100 m (328 ft) buffer strip between the application area and a first-order perennial stream reduced picloram concentrations down to sporadic peaks of less than 0.010 ppm during 17 months of monitoring.

Picloram pellets were also applied to an Upper Coastal Plain site in Alabama. On that watershed, picloram was applied at a slightly higher rate by air (Michael and others 1987). Buffer strips for perennial streams were established but demarcation difficulties resulted in some of the stream areas being overflown. Streamflow at site of the overflight contained a maximum of 0.241 ppm. Picloram concentrations downstream were diluted down to a maximum of 0.077 ppm but persisted for over 475 days in the 0.020 to 0.030 ppm range.

SULFOMETURON METHYL: This herbicide belongs to the substituted-urea class of chemicals. It is very low in toxicity to aquatic and terrestrial organisms. Sulfometuron methyl is readily absorbed and translocated by roots and foliage. Its solubility in the soil is pH dependent, decreasing as acidity rises. Hydrolysis and microbial metabolism are the major degradation pathways which produce a short half-life (Anderson and Dulka 1985). Sulfometuron methyl is available in two formulations, and applied at very low rates (approximately 0.2 kg/ha or 4 oz/ac).

Sulfometuron methyl is a fairly new herbicide. Its environmental fate has been studied at two sites in the South (Michael and Neary 1987). The herbicide was applied as water-dispersible granules and pellets to large (450 ha) watersheds in Mississippi, and small (4 ha) watersheds in Florida. A 15-m (49 ft) streamside buffer strip was used in the Mississippi study and a 5-m (16 ft) one in Florida. At both sites, residues of this herbicide in streamflow were intermittent and did not persist beyond 7 days (Florida) to 63 days (Mississippi). The long persistence in Mississippi was attributed to low soil temperatures at the time of application which slowed hydrolysis and microbial degradation. Most movement of sulfometuron methyl occurred during the first two storms and the herbicide was not detectable beyond 150 m downstream. The peak concentrations were very low at 0.007 ppm (Florida) and 0.044 ppm (Mississippi).

TEBUTHIURON: Tebuthiuron is another herbicide belonging to the substituted-urea group. Unlike sulfometuron methyl, it has a very long half-life (table 1) and is more strongly adsorbed in the soil. Tebuthiuron accumulates in plants where it is subject to metabolic breakdown. It leaches slowly in the soil due to its lower solubility and its adsorption tendency. In water, tebuthiuron does not hydrolyze, and photodegradation losses are negligible.

The movement of tebuthiuron in surface water has been studied mainly on grasslands of the southwest. Its transport and appearance in streamflow is a function of sediment movement since the herbicide is strongly adsorbed onto soil surfaces. A study of tebuthiuron movement (Sassman and Jacobs 1986) reported a peak streamflow concentration of 0.180 ppm from a 2.2 kg/ha (2.0 lb/ac) application, but residues were still detectable at low levels (0.007 ppm) 2 years later. Bovey and others (1978) simulated rainfall on small plots and produced a runoff concentration of 2.230 ppm after application of the same rate of tebuthiuron. However, after 3 months concentrations were down to 0.040 ppm and were not detectable after 13 months. Emmerich and others (1984) reported low amounts of tebuthiuron loss (less than 0.5 percent) from rangelands in Arizona. There are no data available on tebuthiuron movement elsewhere in forested watersheds.

TRICLOPYR: This herbicide is a picolinic acid compound available in amine salt or ester formulations. It is readily absorbed by roots and foliage and translocates easily to meristems. Triclopyr is metabolized by bacteria and photodegrades rapidly. Its half life is less than 10 hours in water but it is more persistent in soils (table 1). It is moderately soluble and not strongly adsorbed in the soil, but studies indicate that it should not be a leaching problem under normal use (Lee and others 1986). Triclopyr, like most of the other forestry herbicides, is low in toxicity to wildlife and fish.

In a West Virginia study, triclopyr applied at a rate of 11.2 kg/ha (10 lb/ac) to small watersheds resulted in peak streamflow concentrations of only 0.080 ppm (McKellar and others 1982). Triclopyr was applied to small watersheds (4 ha or 10 ac) in Florida in both the amine (2.0 kg/ha or 1.8 lb/ac) and ester (1.6 kg/ha or 1.4 lb/ac) formulations. Buffers of 5 m were left next to ephemeral stream channels. Monitoring of streamflow for 5 months following application did not detect any residues of triclopyr (Neary and others 1987).

HERBICIDE RESIDUES IN GROUND WATER

Contamination of ground water has become a national priority environmental issue in the past few years because of growing incidents of herbicide residues being detected in wells. In most of the South, rural residences depend on ground water for a water supply. Also, significant areas of the Coastal Plain utilize ground water for major municipal water sources. For the region as a whole, 98 to 100 percent of the rural population relies on ground water while 14 to 89 percent of the urban population does (Canter and others 1987). Thus it is important to address the issue of potential ground water pollution from operational use of forestry herbicides.

In general, forestry herbicides pose a low pollution risk to ground water because of their use pattern. Herbicide use in forestry is only 10 percent of agricultural usage and likely to occur only once or twice in rotations of 30 to 100 years. Application rates are generally low (less than 2 kg/ha) and animal toxicities are low. Some of the silvicultural herbicides can affect non-target plants at low concentrations (less than 0.020 ppm) and could affect water quality for irrigation. Within large watersheds where extensive ground water recharge occurs, intensive use of silvilcultural herbicides would occur in a dispersed pattern on less than 5% of the area in any one year. Thus the potential for dilution of herbicide residues is enormous.

Regional, confined ground water aquifers are not likely to be affected by forestry herbicides (Neary 1985a). Unconfined surface aquifers in the immediate vicinity of herbicide application zones have the highest risk of contamination. These aquifers are directly exposed to leaching of residues from the root zone. Discussion will focus on these surface aquifers.

SOURCES: In the operational use of silvicultural herbicides there are two types of sources of herbicide contaminants in ground water. These are point sources which occur as a result of spills in the transportation, storage, mixing, and loading phases of herbicide use. Point source pollution is a hazard with the use of any chemical not just forestry herbicides, and accounts for some of the worst cases of localized ground water pollution. During and after the application of herbicides in forest ecosystems, movement of residues into ground water could occur on a landscape scale. This type of pollution is non-point in nature and will be the focus of this discussion.

The data base on ground water contamination from forestry herbicide use in the South is very limited. Few studies have focused on the non-point source aspect of forestry herbicide fate and transport. Also, because of the infrequent use, and low application rate of forestry herbicides, few aquifer contamination problems have arisen from operational application of forestry herbicides. Some information for typical operational conditions is available for hexazinone, picloram, sulfometuron methyl, triclopyr, and 2,4-D. All of these data are from unconfined surface aquifers within 1 to 6 m (3 to 20 ft) of the soil surface.

2,4-D: This herbicide was hand-sprayed on 9 ha (22 ac) of Watershed 6 at the Coweeta Hydrologic Laboratory in Western North Carolina at a rate of 3.4 kg/ha (3.0 lb/ac). 2,4-D was never detected in baseflow originating from near-surface groundwater perched over consolidated bedrock (Douglass and others 1969). In a recent study on the Chattahoochee National Forest of northern Georgia, 2,4-D was applied by injection to 50 percent of a 3.3 ha (8.0 ac) watershed at a rate of 2.2 kg/ha (2.0 lb/ac). Springlow arising from near-surface groundwater was sampled continuously for 165 days after herbicide application. 2,4-D was not detected in any of the samples collected. A sample collected 300 days after application did not contain 2,4-D (Michael 1985b).

HEXAZINONE: In a study in the Georgia Piedmont, this herbicide was applied in a pellet formulation at a rate of 1.68 kg/ha (1.5 lb/ac) to four small (1ha) first-order watersheds (Neary and others 1983). Subsurface movement of hexazinone in baseflow was detected 3 to 4 months after application of the herbicide during dry weather. Concentrations of ground water entering perennial stream channels were very low (less than 0.024 ppm), and were short in duration (less than 30 days). The peak hexazinone concentration was 25 times lower than one suggested water quality standard for hexazinone (0.600 ppm; Leitch and Flinn 1983), and 20 percent of a published Health Guidance Level for agricultural chemicals in ground water (0.125 ppm; NACA 1985). These hexazinone concentrations were never high enough to adversely impact even the most sensitive aquatic species much less higher organisms (Mayack and others 1982).

In an Arkansas study, hexazinone was applied as a liquid formulation in a spot treatment (2.0 kg/ha) to an 11.5 ha watershed (Bouchard and others 1985). Hexazinone residues were measured consistently in ground water entering perennial stream channels as baseflow for over a year after the application. But concentrations never exceeded 0.014 ppm and were below a suggested water quality standard by a factor of 42.

PICLORAM: Use of this herbicide at low rates (less than 1.0 kg/ha) with 2,4-D for injection has not produced any significant ground water contamination. Monitoring of a number of watersheds in Georgia, Tennessee, and Alabama did not detect picloram residues in baseflow originating from shallow ground water. Application of 5.0 kg/ha (4.5 lb/ac) of picloram as a pelleted formulation for site preparation was monitored in the Appalachian Mountains (Neary and others 1985). Picloram residues were detected in baseflow which fed a spring system of a first-order watershed for only 18 days and were less than 0.001 ppm. Infrequent and short duration pulses of picloram (less than 0.010 ppm) occurred over a 17-month period in a 10 ha (25 ac) first-order perennial stream. Peak concentrations were 1 percent of the suggested drinking water standard, but close to levels which might affect sensitive agricultural crops. In-channel dilutions between the treated watershed and any potential irrigation intakes were of such a large magnitude to preclude deterioration in irrigation water quality.

SULFOMETURON METHYL: A study of sulfometuron methyl impact on shallow ground water was recently completed in the Coastal Plain of north Florida (Michael and Neary 1987). Application of 0.42 kg/ha (0.37 lb/ac) active ingredient by ground spray and granule spreading systems to two flatwoods watersheds did not affect ground water quality. Samples were collected from 14 wells for a year. Water in this highly sensitive ground water system (less than 1 m below the ground surface) never contained detected herbicide residues. The rate of sulfometuron methyl application was relatively low compared to other forestry herbicides, but high for this particular chemical.

TRICLOPYR: Another study of herbicide residue fate and movement into shallow ground water was conducted in the Coastal Plain flatwoods. Triclopyr was applied to small watersheds (4 ha - 10 ac) in both the amine (2.0 kg/ha or 1.8 lb/ac) and ester (1.6 kg/ha or 1.4 lb/ac) formulations. Monitoring of 14 surface ground water wells for 5 months following application did not detect any residues of triclopyr (Neary and others 1987).

OTHER HERBICIDES: Ground water data on the other herbicides analyzed in this environmental impact statement are not available for typical forestry situations. Additional research is planned to fill these data gaps. The topic of ground water contamination by pesticides has become a national priority research issue. Most problems have resulted from repeated applications of agricultural pesticides. Based on the limited forestry data, which include a very soluble chemical applied at a high rate (picloram), normal use of the other herbicides should not pose a ground water contamination problem much less a pollution one.

SUMMARY AND CONCLUSIONS

This paper has examined the patterns and types of herbicides used on national forests in the South. These forest ecosystems often overlie major ground water recharge zones and contain streams often used for domestic water supplies. We have discussed the limited herbicide fate and movement data from typical forestry uses to indicate some of the potential impacts on water quality. The following conclusions can be made regarding the impacts of silvicultural herbicides on the quality of surface waters and ground water:

1. The majority of herbicide use in intensive forestry will involve low-toxicity chemicals applied infrequently (once or twice in 30- to 100-year rotations) over extensive land areas.

2. Current herbicide application technology exists to minimize herbicide residue movement into sensitive surface waters. Short-duration residue concentrations of 0.5 to 1.0 ppm might occur during stormflow. On-site degradation processes and in-stream dilution and degradation result in quick dissipation of herbicide residues. Short-term water quality effects are minimal, and long-term water quality is not adversely affected. Long-term water quality can be improved by herbicide use since stream sedimentation is reduced.

3. Site productivity in Southern forests can be increased significantly by herbicide use. There is no documentation or indication of adverse biological effects from use of the silvicultural chemicals examined in this environmental impact statement.

4. At currently registered herbicide application rates, some short duration, low level (less than 0.024 ppm) pulses of herbicide residues could enter unconfined surface aquifers. Detectable residues would not persist for a long time and would not be likely to exceed water quality standards. Contamination of regional ground water aquifers is not likely with even intensive operational use of silvicultural herbicides.

5. The greatest hazards to surface and ground water quality arise from a possible accident or mishandling of concentrates during transportation, storage, mixing and loading, equipment cleaning, and container disposal phases of the herbicide use cycle.

LITERATURE CITED

Anderson, H. W.; Hoover, M. D., Reinhart, K. G. 1976. Forest and water: effects of forest management on floods, sedimentation, and water supply. Gen. Tech. Rep. PSW-18. Davis, CA: U. S. Dep. Agric., For. Serv., Pac. Southwest. For. & Range Exp. Stn. 115 p.

Anderson, J. J.; Dulka, J. J. 1985. Environmental fate of sulfometuron methyl in aerobic soils. J. Agric. Food Chem. 33: 597-602.

Bacon, C. G.; Zedaker, S. M. 1987. Third-year growth response of loblolly pine to eight levels of competition control. South. J. Appl. For. 11: 91-95.

Baur, J. R.; Bovey, R. W.; Merkle, M. G. 1972. Concentration of picloram in runoff water. Weed Sci. 20: 309-313.

Beasley, R. S. 1979. Intensive site preparation and sediment loss on steep watersheds in the Gulf Coastal Plain. Soil Sci. Soc. Am. Proc. 43: 412-417.

Beasley, R. S.; Granillo, A. B.; Zillmer, V. 1986. Sediment losses from forest management: mechanical vs. chemical site preparation after clearcutting. J. Env. Qual. 15: 413-416.

Bouchard, D. C.; Lavy, J. L.; Lawson, E. R. 1985. Mobility and persistence of hexazinone in a forested watershed. J. Env. Qual. 14: 229-233.

Bovey, R. W.; Burnett, E.; Meyer, R. E.; Richardson, C.; Lon, A. 1978. Persistence of tebuthiuron in surface water runoff water, soil, and vegetation in the Texas Blacklands Prairie. J. Environ. Qual. 7: 233-236.

Canter, L. W.; Knox, R. C.; Fairchild, D. M. 1987. Ground water quality protection. Chelsea, MI: Lewis Publ., Inc. 562 p.

Cantrell, R. L. 1985. A guide to silvicultural herbicide use in the southern United States. Auburn, AL: Auburn Univ. Sch. For. 575 p.

Crossley, D. A.; Swank, W. T., eds. 1987. Forest hydrology and ecology at Coweeta. New York, NY: Springer-Verlag. 469 p.

Douglass, J. E.; Cochrane, D. R.; Bailey, G. W.; Teasley, J. I.; Hill, D. W. 1969. Low herbicide concentration found in streamflow after a grass cover is killed. Res. Note SE-108. Asheville, NC: U. S. Dep. Agric., For. Serv., Southeast. For. Exp. Stn. 3 p.

Douglass, J. E.; Goodwin, O. C. 1980. Runoff and soil erosion from forest site preparation practices. In: U. S. Forestry and water quality: what course in the 1980s? Richmond, VA: Water Pollut. Control Fed. 22 p.

Douglass, J. E.; Van Lear, D. H. 1983. Prescribed burning and water quality of ephemeral streams in the piedmont of South Carolina. For. Sci. 29: 181-189.

Eijasackers, H.; Bund, C. F. van de. 1980. Chapter 10: Effects on soil fauna. In: Hance, R. J., ed. 1980. Interactions between herbicides and the soil. New York, NY: Academic Press; 255-305.

Emmerich, W. E.; Helmer, J. D.; Renard, K. G.; Lane, J. L. 1984. Fate and effectiveness of tebuthiuron applied to a rangeland watershed. J. Environ. Qual. 13: 382-386.

Fletcher, K.; Friedman, B. 1986. Effects of the herbicides glyphosate, 2,4,5-T, and 2,4-D on forest litter decomposition. Can. J. For. Res. 16: 6-9

Glover, G. R. 1985. Growth response of southern pines to control of competing vegetation. In: Pywell, N. A.; Neary, D. G.; Law, B., eds. 1985. Herbicides for southern forestry, proceedings of the 1985 annual spring symposium, School of Forest Resources and Conservation. Gainesville, GL: Univ. Fla; 212-222.

Greaves, M. P.; Davies, H. A.; Marsh, J. A. P.; Wingfield, G. I. 1976. Herbicides and soil micro-organisms. CRC Critical Rev. Microbiol. 5: 1-38.

Greaves, M. P.; Malkoney, H. P. 1980. Chapter 9: Effects soil microflora. In: Hance, R. J., ed. 1980. Interactions between herbicides and the soil. New York, NY: Academic Press; 223-253.

Grover, R., ed. 1988. Environmental chemistry of herbicides. Boca Raton, FL: CRC Press. 208 p.

Hance, R. J., ed. 1980. Interactions between herbicides and the soil. New York, NY: Academic Press. 349 p.

Hewlett, J. R. 1982. Principles of forest hydrology. Athens, GA: Univ. Ga. Press. 183 p.

Kellison, R. C.; Gingrich, S. 1982. Proceedings of the symposium on the loblolly pine ecosystem (East region); December 8-10, 1982; Raleigh, NC. Raleigh, NC: N. C. State Univ., Sch. For. Resour. 335 p.

Knowe, S. A.; Nelson, L. R.; Gjerstad, D. H.; Zutter, B. R.; Glover, G. R.; Minogue, P. J.; Dukes, J. H., Jr. 1985. Four year growth and development of planted loblolly pine on sites with competition control. South. J. Appl. For. 9: 11-15.

Larsen, W. E.; Pierce, F. J.; Dowdy, R. H. 1983. The threat of soil erosion to long-term crop production. Science 219: 458-465.

Lee, C. H.; Oloffs, P. C.; Szeto, S. Y. 1986. Persistence, degradation, and movement of triclopyr and its ethylene glycol butyl ester in a forest soil. J. Agric. Food Chem. 34: 1075-1079.

Leitch, C. J.; Flinn, D. W. 1983. Residues of hexazinone in stream wash after aerial application to an experimental catchment planted with radiata pine. Austr. For. 46: 126-131.

Martin, J. P. 1963. Influence of pesticide residues on soil microbiological and chemical properties. Residue Rev. 4: 98-129.

Mayack, D. T.; Bush, P. B.; Neary, D. G.; Douglass, J. E. 1982. Impact of hexazinone on invetebrates after application to forested watersheds. Arch. Env. Contam. Toxicol. 11: 209-217.

McKellar, R. L.; Schubert, O. E.; Byrd, B. C.; Stevens, L. P.; Norton, E. J. 1982. Aerial application of Garlon 3A herbicide to a West Virginia watershed. Down to Earth 38(2): 15-19.

Michael, J. L. 1980. Long-term impact of aerial application of 2,4,5-T to longleaf pine. Weed Sci. 28: 255-257.

Michael, J. L. 1985a. Growth of loblolly pine treated with hexazinone, sulfometuron methyl, and metsulfuron methyl for herbaceous weed control. South. J. Appl. For. 9: 20-26.

Michael, J. L. 1985b. Movement and non-target impacts of hexazinone and picloram on inject sites: hexazinone and tordon 101 from 1983 application on the Cohutta Ranger District on the Chattahoochee National Forest. Prog. Rep. FS-SO-1116-1.16 prob. 1. Washington, DC: U. S. Dept. Agric., For. Serv., NAPIAP.

Michael, J. L. 1986. Fate of Arsenal in forest watersheds after aerial application for forest weed control. Final Rep. Auburn, AL: U. S. Dep. Agric., For. Serv., South. For. Exp. Stn.

Michael, J. L.; Neary, D. G. 1987. Movement of sulfometuron methyl in forest watersheds after aerial application of Oust for herbaceous weed control. Final Rep. Auburn, AL: U. S. Dep. Agric., For. Serv., South. For. Exp. Stn. 39 p.

Michael, J. L.; Neary, D. G.; Wells, M. J. M. 1987. Picloram movement in soil solution and streamflow from a coastal plain forest. J. Environ. Qual. (submitted)

Miller, J. H.; Bace, A. C. 1980. Streamwater contamination after aerial application of pelletized herbicide. Res. Note SO-255. New Orleans, LA: U. S. Dep. Agric., For. Serv., South. For. Exp. Stn. 4 p.

Miller, J. H.; Williamson, M. 1987. Weeds in your weedlot? Amer. Tree Farmer 3: 8-9.

National Research Council. 1983. Drinking water and health, Vol. 5. Washington, DC: National Academy Press.

National Research Council of Canada. 1974. Picloram: The effects of its use as a herbicide on environmental quality. Publ. 13684. Ottawa, Ontario, Can.: Nat. Res. Counc. Can. 128 p.

Neary, D. G. 1983. Monitoring herbicide residues in springflow after an operational application of hexazinone. South. J. Appl. For. 7: 217-223.

Neary, D. G. 1985a. Fate of pesticides in Florida's forests: an overview of potential impacts on water quality. Soil Crop Sci. Soc. Fla. Proc. 44: 18-23.

Neary, D. G. 1985b. New and old herbicides for weed control in Florida's forests--an introduction. In: Pywell, N. A.; Neary, D. G.; Law, B. 1985. Proceedings of the 1985 spring symposium on herbicides for southern forestry; April 23-24, 1985; Gainesville, FL. Gainesville, FL: Univ. Fla., Sch. For. Resour. & Conserv.; 1-15.

Neary, D. G. 1987. Hydrologic impacts of forest pesticide use. In: Carpenter, S. B. 1987. Proceedings: southern forestry symposium; November 19-21, 1985; Atlanta, GA. Atlanta, GA: Nat. Assoc. Prof. For. Sch. & Coll., South. Reg.; 288-310.

Neary, D. G.; Bush, P. B.; Douglass, J. E. 1983. Offsite movement of hexazinone in stormflow and baseflow from forest watersheds. Weed Sci. 31: 543-551.

Neary, D. G.; Bush, P. B.; Douglass, J. E.; Todd, R. L. 1985. Picloram movement in an Appalachian hardwood forest watershed. J. Env. Qual. 14: 585-592.

Neary, D. G.; Bush, P. B.; Grant, M. A. 1986. Water quality of ephemeral forest streams after site preparation with the herbicide hexazinone. For. Ecol. Manage. 14: 23-40.

Neary, D. G.; Bush, P. B.; Taylor, J. W., Jr. 1987. Triclopyr fate and movement in a coastal plain flatwoods forest ecosystem. Prog. Rep. Gainesville, FL: U. S. Dep. Agric., For. Serv., Southeast. For. Exp. Stn. 6 p.

Neary, D. G.; Morris, L. A.; Swindel, B. F. 1984. Site preparation and nutrient management in southern pine forests. In: Stone, E. L., ed. Forest soils and treatment impacts: proceedings, 6th North American forest soils conference; June 1983; Knoxville, TN. Knoxville, TN; Univ. Tenn.; 121-144.

Neary, D. G.; Rockwood, D. L.; Comerford, N. B.; Swindel, B. F.; Cooksey, T. C. [1988]. Importance of water, nutrients, and genetics in slash and loblolly pine growth potential on poorly-drained spodosols. For. Ecol. & Manage. (In press).

Neary, D. G.; Taylor, J. W. 1984. Herbicide use: safety, environmental, and political considerations in forestry. In: Kurt, F. R., coord. Proceedings: 4th annual forestry forum: herbicides: prescription and application; March 21, 1984; Clemson, SC. Clemson, SC: Coop. Extens. Serv.; 45-65.

Nelson, L. R.; Peterson, R. C.; Autry, C. L.; Dudley, S.; Walstad, J. D. 1981. Impacts of herbaceous weeds in young loblolly pine plantations. South. J. Appl. For. 5: 153-158.

Newton, M.; Howard, K. M.; Kelpsas, B. R.; Danhaus, R.; Lottman, C. M.; Dubleman, S. 1984. Fate of glyphosate in an Oregon forest ecosystem. J. Agric. Food Chem. 32: 1144-1151.

Norris, L. A. 1981a. The behavior of herbicides in the forest environment and risk assessment. In: Holt, H. H.; Fischer, B. C., eds. 1981. Weed control in forest management; Feb. 3-5, 1981; Champaign-Urbana, IL. Champaign-Urbana, IL: Purdue Univ.; 192-216.

Norris, L. A. 1981b. The movement, persistence, and fate of phenoxy herbicides and TCDD in the forest. Res. Rev. 80: 65-135.

Norris, L. A.; Montgomery, M. L. 1975. Dicamba residues in streams after forest spraying. Bull. Environ. Contam. Toxicol. 13: 1-8.

Rhodes, R. C. 1980. Soil studies with c14 labelled hexazinone. J. Agric. Food Chem. 28: 311-315.

Rueppel, M. L.; Brightwell, B. B.; Schafer, J.; Marvel, J. T. 1977. Metabolism and degradation of glyphosate in soil and water. J. Agric. Food Chem. 25: 517-523.

Sacher, R. M. 1978. Safety of Roundup in the aquatic environment. In: Proceedings of the EWRS 5th symposium on aquatic weeds. Amsterdam; 315-322.

Sassman, J. F.; Jacobs, M. M. 1986. Pesticides background statements. Vol. I. Herbicides (supplement). Agric. Handb. 633. Washington, DC: U. S. Dep. Agric., For. Serv. 88 p.

Sassman, J. F.; Pienta, R.; Jacobs, M.; Cioffi, J. 1984. Pesticides background statements. Vol. I. Herbicides. Agric. Handb. 633. Washington, DC: U. S. Dep. Agric., For. Serv. 1075 p.

Scifres, C. J.; Allen, T. J.; Leinweber, C. L.; Pearson, K. H. 1973. Dissipation and phytotoxicity of dicamba residues in water. J. Environ. Qual. 2: 360-309.

Singer, J. 1980. Pesticide safety guidelines for personnel protection. Unnumb. publ. Davis, CA: U. S. Dep. Agric., For. Serv., For. Ins. & Disease Manage., Meth. Appl. Group. 45 p.

Smith, A. E.; Cullimore, D. R. 1975. Microbiological degradation of the herbicide dicamba in moist soils at different temperatures. Weed Res. 15: 59-62

Stone, E. L. 1983. The managed slash pine ecosystem. Unnumb. Publ. Gainesville, FL: Univ. Fla., Sch. For. Resour. & Conserv. 434 p.

Swindel, B. F.; Neary. D. G.; Comerford, N. B.; Rockwood, D. L.; Blakeslee, G. M. Fertilization and competition control accelerate early southern pine growth. South. J. Appl. For. (In press).

United States Department of Agriculture and Environmental Protection Agency. 1975. Apply pesticides correctly. Washington, DC: U. S. Gov. Print. Ofc. 26 p.

Weed Science Society of America. 1986. Herbicide handbook, 6th Ed. Champaign, IL: Weed Sci. Soc. Am. 515 p.

APPENDIX D

A Biological Evaluation of the Effects of the Preferred Alternative on Threatened, Endangered, Proposed, and Sensitive Species

APPENDIX D

A BIOLOGICAL EVALUATION OF THE EFFECTS OF THE FINAL PREFERRED ALTERNATIVE ON THREATENED, ENDANGERED, PROPOSED, AND SENSITIVE SPECIES

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Summary

This biological evaluation addresses the effects of vegetation management activities described in the Final Environmental Impact Statement for Vegetation Management in the Ozark/Ouachita Mountains for the Southern Region of the U. S. Forest Service on threatened, endangered, proposed, and sensitive species. Effects of the program were determined to be beneficial or not detrimental. For threatened, endangered, and proposed species, concurrence from the USDI Fish and Wildlife Service is recommended. For sensitive species, informal coordination between affected national forests and appropriate State heritage agencies is recommended.

Introduction

Objectives:

The objectives of this biological evaluation are to:

1. Determine the effects of the program of vegetation management activities identified in the preferred alternative on threatened, endangered, and proposed plant and animal species occurring in national forests in the Ozark/Ouachita Mountains.

2. Determine the effects of the program of vegetation management activities identified in the preferred alternative on sensitive plant and animal species occurring in national forests in the Ozark/Ouachita Mountains.

3. Describe measures to be taken to mitigate potential adverse effects of activities described in the preferred alternative on threatened, endangered, or proposed species.

4. Describe measures to be taken to mitigate potential adverse effects of activities described in the preferred alternative on sensitive species.

This biological evaluation was prepared in accordance with Forest Service Manual 2671.44 and 2672.43 and regulations set forth in section 7(a) of the Endangered Species Act (ESA).

Nine animal species classified by the U. S. Fish and Wildlife Service as threatened or endangered (or proposed for listing as threatened or endangered) live in the Ozark and Ouachita National Forests. These species include two mammals, four birds, one reptile, two mollusks, and one land snail. There are no threatened or endangered plant species on either forest. Habitats of these species are managed under authority of the Endangered Species Act with the goal of population recovery.

In addition, certain species for which population viability is a concern are designated by the Regional Forester as "sensitive." Normally, this designation is established with the concurrence and guidance of the appropriate State Heritage Agency. Species listed in tables E-3 and E-4 include species so designated at the time this appendix was prepared and species being reviewed by the U. S. Fish and Wildlife Service for possible addition to the List of Endangered and Threatened Species under the Endangered Species Act of 1973, as amended, and describes their habitats. Habitats of sensitive species are managed to ensure population levels which will keep these plants and animals from becoming threatened or endangered.

Evaluation Methods

This evaluation was conducted from January 23 - February 21, 1989 and is based upon:

1. Review of FSH 2609.23R-R8 AMEND. (The Wildlife Habitat Management Handbook) chapters 418, 420, 421, 422.

2. Review of recovery plans for the southern bald eagle, red-cockaded woodpecker, gray bat, Indiana bat, and American alligator.

3. Review of the scientific literature related to effects of vegetation management on listed species, including the following references:

Ajilvsgi, G. 1979. Wildflowers of the Big Thicket, East Texas, and Western Louisiana. College Station, TX: Texas A&M Univ. Press. 360 p.

Arkansas Department of Planning. 1974. Arkansas natural area plan. 248 p.

Borror, D. J.; White, R. E. 1970. A field guide to the insects. Boston, MA: Houghton Mifflin Co. 152 p.

Burnside, F. L. 1987. Long-distance movements by loggerhead shrikes. Journal of Field Ornithology 58(1): 62-65.

Burnside, F. L.; Shepherd, W. M. 1985. Population trends of the loggerhead shrike (*Lanius ludovicianus*) in Arkansas. Proc. Ark,. Acad. Sci., 39:25-28.

Burt, W. H.; Grossenheider, R. P. 1976. A field guide to the mammals. Boston, MA: Houghton Mifflin Co. 289 p.

Caire, W. 1985. Summer ecology of the bats in southeastern Oklahoma. Final Report to the Oklahoma Department of Wildlife Conservation - Nongame Division, Oklahoma City, 22 p.

Chamberlain, E. B. 1974. Rare & endangered birds of the southern national forests. Atlanta, GA: U. S. Dep. Agric., For. Serv., South. Reg. 108 p.

Clewell, A. F. 1985. Guide to the vascular plants of the Florida Panhandle. Gainesville, FL: Univ. Presses Fla. 605 p.

Collins, J. J., Jr. 1981. Harper & Row's complete field guide to North American wildlife--eastern edition. New York, NY: Harper & Row Publ., Inc.

Collins, H. H., Jr. 1959. Complete field guide to North American wildlife. New York, NY: Harper & Row Publ., Inc. 714 p.

Conant, R. 1975. A field guide to reptiles and amphibians of eastern and central North America; second edition. Boston, MA: Houghton Mifflin Co. 429 p.

Cooper, J. E.; Robinson, S. S.; Funderburg, J. B. 1977. Endangered and threatened plants and animals of North Carolina: proceedings of the symposium on endangerd and threatened biota of North Carolina. 1. Biological concerns. Raleigh, NC: N. C. State Mus. Nat. Hist. 444 p.

Correll, D. S.; Johnston, M. C. 1970. Manual of the vascular plants of Texas. Renner, TX: Tex. Res. Found. 1881 p.

Davis, R. H.; Rettig, J. H. 1984. Rare plants. Pp. 36-47. In Arkansas's natural heritage. Bill Shepherd, editor. August House Publishers, Little Rock, Ar., 116 p.

Davis, W. H.; Lidicker, W. Z.; Sealander, J. A. 1955. *Myotis austroriparius* in Arkansas. Journal of Mammalogy 36:288.

Fernald, M. L. 1970. Gray's manual of botany. New York, NY: B. Van Nostrand Co. 1632 p.

Glass, B. P.; Ward, C. M. 1959. Bats of the genus *Myotis* from Oklahoma. Journal of Mammalogy 40(2):194-201.

Harris, J. L.; Gordon, M. E. 1987. Distribution and status of rare and endnagered mussels (Mollusca: Margaritiferidae, Unionidae) in Arkansas. Proc. Ark. Acad. Sco., 41:49-56.

Harvey, M. J.; Cassidy, J. J.; O'Hagan, G. G. 1981. Endangered bats of Arkansas: distribution, status, ecology, and management. Report to the Arkansas Game and Fish Commission, USFS, Ozark National Forest, and USDI National Park Service, Buffalo National Park. Ecological Research Center, Memphis State University, TN, 80 p.

Heath, D. R.; Saugey, D. A.; Heidt, G. A. 1986. Abandoned mine fauna of the Ouachita Mountains, Arkansas: vertebrate taxa. Proc. Ark. Acad. Sci. 40:33-36.

Hunter, C. G. 1984. Wildflowers of Arkansas. The Ozark Society Foundation, Little Rock, Ar., 296 p.

James, D. A.; Neal, J. C. 1986. Arkansas birds; their distribution and abundance. The University of Arkansas Press, Fayetteville, 402 p.

Kral, R. 1983. A report on some rare threatened, or endangered forest-related vascular plants of the South. Volume I. Isoetaceae through Euphorbiaceae. Tech. Publ. R8-TP 2. Atlanta, GA: U. S. Dep. Agric., For. Serv., South. Reg. 718 p.

Kral, R. 1983. A report on some rare threatened, or endangered forest-related vascular plants of the South. Volume II. Aquifoliacea through Asteraceae and Glossary. Tech. Publ. R8-TP 2. Atlanta, GA: U. S. Dep. Agric. For. Serv., South. Reg. 586 p.

Lechner, M.; Zale, A. V.; O'Donnell, S.; Ben, B. 1987. Distribution and abundances of the leopard darter, *Percina pantherina* (Moore and Reeves), in the upper Mountain Fork and Little River Drainages. Report to the Tulsa District, U. S. Army Corps of Engineers. Prepared by the Oklahoma Cooperative Fish and Wildlife Unit, Oklahoma Cooperative Fish and Wildlife Unit, Oklahoma State University, Stillwater, 27 p.

Lee, D. S.; Gilbert, C. R.; Hocutt, C. H.; Jenkins, R. E.; McAllister, D. E.; Stauffer, J. R., Jr. 1980. Atlas of North American freshwater fishes. Raleigh, NC: N. C. Dep. Agric., N. C. State Mus. 854 p.

Leon, S. C.; Ferguson, M. T.; Maughn, O. E.; Zale, A. V. 1987. Distribution and abundance of the leopard darter, *Percina pantherina* (Moore and Reeves). Report to the Tulsa District, U. S. Army Corps of Engineers by the Oklahoma Cooperative Fish and Wildlife Unit, Oklahoma State University, Stillwater. 66 p.

Lowman, G. E. 1975. A survey of endangered, threatened, rare, status undertermined, peripheral, and unique mammals of the southeastern national forests and grasslands. Unnumb. Publ. [Contr. 38-2601]. Atlanta, GA: U. S. Dep. Agric., For. Serv., South. Reg. 121 p.

Lowery, G. H., Jr. 1974. The mammals of Louisiana and its adjacent waters. Baton Rouge, LA: La. State Univ. Press. 565 p.

Martof, B. S., et al. 1980. Amphibians and reptiles of the Carolinas and Virginia. Chapel Hill, NC: Univ. N. C. Press.

McDaniel, V. R.; Gardner, J. E. 1977. Cave fauna of Arkansas: Vertebrate taxa. Proc. Ark. Ark. Acad. Sci. 31:68-71.

Odom, R. R.; McCollum, J. L.; Nevill, M. A.; Ettman, D. R. 1977. Georgia's protected wildlife. Social Circle, GA: Ga. Dep. Nat. Resour., Game & Fish Div., Endangered Wildl. Prog. 51 p.

Oklahoma Department of Wildlife Conservation. 1985. Oklahoma's endangered species. Oklahoma City.

Peterson, R. T. 1947. A field guide to the birds. Boston, MA: Houghton Mifflin Co. 230 p.

Peterson, R. T. 1980. A field guide to the birds east of the Rockies; fourth edition. Boston, MA: Houghton Mifflin Co. 384 p.

Plummer, M. V. 1982. The status of the Caddo Mountain and Fourche Mountain salamanders (*Plethodon caddoensis*, *P. fourchensis*) in Arkansas. A report prepared for the Arkansas Natural Heritage Commission, Little Rock. 12 p.

Porcher, R. D. 1980. Inventory of populations of proposed endangered and threatened species of vascular plants of the Francis Marion National Forest in Berkeley and Charleston Counties, South Carolina. Fin. Rep. [Contr. 53-43ZP-8-00008]. Atlanta, GA: U. S. Dep. Agric., For. Serv., South. Reg. 101 p.

Pritchard, P. C. H., ser. ed. [1979.] Rare and endangered biota of Florida. Vols. 1 - 5. Gainesville, FL: Univ. Presses Fla. 480 p. [Vol. 1 -- Layne, J. N., ed. Mammals; Vol. 2 -- Kale, J. W., II, ed. Birds; Vol. 3 -- McDiarmid, R. W., ed. Amphibians and reptiles; Vol. 4 -- Gilbert, C. R., ed. Fishes; Vol. 5 -- Ward, D. B., ed. Plants.]

Pulliam, J. J. 1987. Site summary, Rich Mountain, Polk County, Arkansas. [Species concerned: Rich Mountain slitmounth snail (*Stenotrema pilsbryi*), mountain cave amphipod (*Stygobromus montanus*)]. U. S. Fish and Wildlife Service, Jackson, MS. 1 p.

Radford, A. E.; Ahles, H. E.; Bell, C. R. 1968. Manual of the vascular flora of the Carolinas. Chapel Hill, NC: Univ. N. C. Press. 1183 p.

Robinson, A. F., Jr. n.d. Possible impacts of silvicultural activities on proposed endangered and threatened plant species of pine flatwoods. Atlanta, GA: U. S. Dep. Agric., For. Serv., South. Reg.

Robison, H. W. 1978. The leopard darter (a status report). Endangered Species Report No. 3. Prepared for the U. S. Fish and Wildlife Service, Albuquerque, New Mexico. 28 p.

Robison, H. W.; Buchanan, T. M. 1988. Fishes of Arkansas. The University of Arkansas Press, Fayetteville. 536 p.

Robison, H. W.; Smith, K. L. 1982. The endemic flora and fauna of Arkansas. Proc. Ark. Acad. Sci., 36:52-57.

Robison, H. W.; Smith, K. L. 1984. Aquatic ecosystems. Pp. 97-113. In Arkansas's natural heritage. Bill Shepherd, editor, August House Publishers, Little Rock, Ar. 116 p.

Roedner, B. J.; Hamilton, D. A.; Evans, K. E. 1978. Rare plants of the Ozark Plateau - a field identification guide. USDA Forest Service, North Central Forest Experiment Station, St. Paul, MN. 238 p.

Saugey, D. A.; Heidt, G. A.; Heath, D. R. 1988. Utilization of abandoned mine drifts and fracture caves by bats and salamanders: unique subterranean habitat in the Ouachita Mountains. Proceedings Symposium on the Management of amphibians, reptiles, and small mammals in North America, Flagstaff, AZ, In press.

Saugey, D. A.; Heidt, G. A.; Heath, D. R. 1989. Survey of bats in the Ouachita Mountains. Manuscript submitted to Proc. Ark. Acad. Sci.

Sealander, J. A. 1979. A guide to Arkansas mammals. River Road Press, Conway, Ar. 313 p.

Seehorn, M. E. 1982. Reptiles and amphibians of southeastern national forests. Atlanta, GA: U. S. Dep. Agric., For. Serv., South. Reg. 85 p.

Sievert, G. 1986. An investigation of the distribution and population status of the Rich Mountain salamander (Plethodon ouachitae) in Oklahoma. A report prepared for the Oklahoma Department of Wildlife Conservation - Nongame Division, Oklahoma City. 37 p.

Sievert, G.; Sievert, L. 1988. A field guide to reptiles of Oklahoma. Oklahoma Department of Wildlife Conservation, Oklahoma City. 96 p.

Small, J. K. 1933. Manual of the southeastern flora. Chapel Hill, NC: Univ. N. C. Press. 1554 p.

Smith, K. L. 1984. Animal species. Pp. 48-70. In Arkansas's natural heritage. Bill Shepherd, editor. August house Publishers, Little Rock. Ar. 116 p.

Southeastern Wildlife Services. 1980. Inventory of threatened or endangered plants on the Sumter National Forest. Columbia, SC: U. S. Dep. Agric., For. Serv., Sumpter Natl. For. 97 p.

Steward, T. W.; McDaniel, V. R.; Saugey, D. A.; England, D. R. 1986. The bat fauna of southwest Arkansas. Proc. Ark. Acad. Sci. 40:72-73.

Steyermark, J. A. 1963. Flora of Missouri. The Iowa State University Press, Ames. 1728 p.

Taylor, W. C. 1984. Arkansas ferns and fern allies. Milwaukee Public Museum, WI. 262 p.

USDI Fish and Wildlife Service. 1987. Status survey of Lampsilis streckeri Frierson (1927) and Arcidens wheeleri (Ortman and Walker, 1912). Final Report from Ecoresearch, Inc., 325 E. Bayview, Portland, TX, 78374.

U. S. Fish & Wildlife Service. 1987. Endangered and threatened species of the southeastern United States. Washington, DC: U. S. Dep. Inter., Fish & Wildl. Serv.

Webster, W. D.; Parnell, J. F.; Biggs, W. C., Jr. 1985. Mammals of the Carolinas, Virginia, and Maryland. Chapel Hill, NC: Univ. N. C. Press. 255 p.

4. Review of relevant sections of the Federal Register.

5. Information presented in the EIS and appendices, including appendix A, the Risk Assessment for the Use of Herbicides in USDA Forest Service Southern Region.

6. Discussions with U. S. Forest Service biologists, botanists, and other specialists:

Mickey Beland
Dennis Danner
Danny Ebert
Ron Escano
Gary Hartman
Larry Hedrick
Jim Herrig
Lauren Hillman
Jimmy Huntley
Ralph Odegard
Levester Pendergrass
Carl Racchini
Ben Sanders
Cecil Thomas
Joan Walker

7. Discussions with other experts:

Regarding distribution and occurrence:

Sam Barkley, Arkansas Game and Fish Commission
Ken Smith, William Shepherd, Arkansas Nat. Heritage Comm.
Dr. John Harris, Arkansas State Highway and Transportation Department

Lance Peacock, Arkansas Nature Conservancy
Drs. V. R. McDaniel, S. E. Trauth, George Harp, Arkansas State University
Dr. William Caire, Central Oklahoma University
Dr. Mike Plummer, Harding University
James E. Gardner, Illinois State Natural History Survey
John Skeen, Oklahoma Department of Wildlife Conservation
Dr. Pat Cifelli, Oklahoma Natural Heritage Inventory
Renn Tumlison, Oklahoma State University
Dr. Henry Robison, Southern Arkansas University
Dr. Michael Harvey, Tennessee Tech University
John Pulliam, U. S. Fish and Wildlife Service
Dr. Douglas James, University of Arkansas at Fayetteville
Drs. G. A. Heidt, Charles Preston, Al Karlin, University of Arkansas, Little Rock
Sue Bozeman, Bill Puckett, Central Oklahoma Grotto, National Speleological Society
Dr. Robert Wilkinson, Southwest Missouri State University
Vernon Bates, Botanist, Altanta, Georgia
Fred Burnside, EPA, Dallas, TX
Dr. Mike Kennedy, Memphis State University

Regarding treatment effects:

Dr. Bert Pittman, Arkansas Nat. Heritage Comm.
Paul Robertson, Bat Conservation International, Austin, TX
Ronald Eisler, USDI, Fish and Wildlife Service
Larry Landers, Tall Timbers Research Station
William McComb, Oregon State University
Phil Sczerzenie, LaBat-Anderson, Inc.
George Hurst, Mississippi State University
Cary Norquist, USDI, Fish and Wildlife Service
Melynda Reid, Volunteer, National Forests in Florida
Dennis Hardin, Florida Natural Areas Inventory
John Palis, Florida Natural Areas Inventory
Paul Hartfield, Mississippi Museum of Natural Science
Joseph Fitzpatrick, Jr. University of South Alabama
Latimore Smith, Louisiana Natural Heritage Program
Nelwyn Gilmore, Louisiana Natural Heritage Program

8. Informal consultation with John Pulliam, U. S. Fish and Wildlife Service, Jackson, Mississippi office, January 1989.

Much of this information was compiled and analyzed by David A. Saugey, Wildlife Biologist, USDA Forest Service.

Indications of adverse or beneficial effect shown in tables D-1, D-3, D-5, and D-6 and based on the best professional opinion of the individuals and sources cited in items 1-8 above. They are not necessarily the result of detailed scientific study and should not be construed as a substitute for site-specific analysis.

Affected Area and Proposed Actions

This evaluation examines the program of vegetation management described in the preferred alternative of the Final EIS. These activities are described in detail in chapter II of the EIS and fall into the broad categories of herbicides, prescribed fire, mechanical, manual, and biological (grazing) methods of managing vegetation. Treatments are employed to accomplish a variety of resource management goals including site preparation for stand establishment, timber and wildlife stand improvement, endangered species habitat management, and rights-of-way (ROW) maintenance. Effects of these treatments on plants and animals are discussed in detail in chapter IV of the EIS.

Proposed activities occur on the Ouachita and Ozark National Forests in two States located in the USDA Forest Service's Southern Region. This area is described in detail in chapter III of the EIS.

Potential Adverse Effects--General Mitigation Measures

As described in mitigation measures detailed in chapter II of the EIS, the following general requirements and measures apply to all vegetation management methods. Each forest may be more restrictive, but not less.

1. All projects will have site-specific analysis, in accordance with the National Environmental Policy Act (NEPA). This environmental analysis will consider site-specific techniques, intensity of application methods, and potential environmental effects of any method considered. A reasonable range of alternative methods, including the use of methods which do not involve herbicides, will be examined and evaluated.

Potential adverse effects on threatened, endangered, and sensitive species will be evaluated.

Requirements and measures for activities affecting threatened, endangered, or proposed species are detailed in species recovery plans and in FSH 2609.23R. Recovery plans have been prepared for the southern bald eagle, red-cockaded woodpecker, gray bat, Indiana bat, American alligator, and fat pocketbook pearly mussel. Chapters in FSH 2609.23R have been prepared for red-cockaded woodpecker, southern bald eagle, and American alligator.

Requirements and measures for activities affecting sensitive species are detailed in Forest Land and Resource Management Plans and amendments.

2. A biological evaluation of how a project may affect any species Federally listed as threatened, endangered, or proposed for listing, or identified by the Forest Service as sensitive, will be conducted as part of the site-specific environmental analysis process.

The site-specific biological evaluation considers all available inventories of threatened, endangered, proposed, and sensitive species populations for the proposed treatment area. When adequate population inventory information is unavailable, it will be collected when the affected site has high potential for occupancy by a threatened, endangered, proposed, or sensitive species. Table D-1 identifies the potential of adverse effects from vegetation management by species. When potential adverse effects are indicated, mitigation measures specified in chapter II of the EIS will be employed to prevent adverse effects.

If it is determined that the project may affect (including beneficial effects) Federally-listed endangered, threatened, or proposed species, consultation is initiated with the U. S. Fish and Wildlife Service. If, during informal consultation, it is determined that the project is not likely to adversely affect listed species and the U. S. Fish and Wildlife Service concurs in writing with that determination, consultation is terminated. However, if it is determined that the project is likely to adversely affect listed species, formal consultation is initiated. Figure D-1 outlines this process.

When the evaluation indicates that a project may have any adverse effect on a species or the habitat of a species listed as sensitive, appropriate State wildlife agencies, natural heritage commissions, and other cooperators or species authorities will be contacted to identify coordination measures. These measures will be directed towards ensuring species viability and preventing negative population trends that would result in Federal listing.

Potential Adverse Effects--Threatened, Endangered, and Proposed Animals

Table D-1 displays general information regarding potential effects of vegetation management methods on endangered, threatened, and proposed animals. The likelihood of adverse effect or significant risk of toxic effects is based on use of vegetation management without mitigation measures.

In most cases, properly applied prescribed fire is beneficial or has no effect on the listed species. However, unless proper techniques are employed, bald eagle nest trees and red-cockaded woodpecker cavity trees may be destroyed. Measures detailed in chapter II of the EIS concerning prescribed burn planning and execution and protection of threatened and endangered species habitat, mitigate this effect. Although effects of burning on the Indiana and gray bats are unknown, it is unlikely that prescribed burning would adversely affect these species since any effect of burning on aerial insect populations (the bats' main food source) in foraging areas would be minimal. Aquatic species are not affected.

Herbicides may be used to improve habitat for the red-cockaded woodpecker. Applied improperly, herbicides may cause the loss of bald eagle nest trees and red-cockaded woodpecker colony trees. These species are protected by restrictions regarding activities near their nesting sites. Habitat for aquatic species, such as the pocketbook pearly mussel, may be degraded if herbicides are used to reduce streamside vegetation. This risk is mitigated by Forest Land and Resource Management Plan standards and guides for protecting aquatic and riparian habitats. There is however, a potential for toxic effects on most threatened and endangered animals when certain herbicides are applied at extreme rates. Table D-2 displays the risk to terrestrial species from broadcast application at normal and extreme rates and to aquatic species from two accidental spill scenarios.

At normal rates, a potential for significant risk (exposure greater than 1/10 LD_{50}) to gray and Indiana bats exists for triclopyr. Measures in chapter II of the EIS regarding selection and application of herbicides, and prohibition of application at extreme rates mitigates risk to animal species except for the gray and Indiana bats. Measures in chapter II restricting the application of triclopyr mitigates risk to these species.

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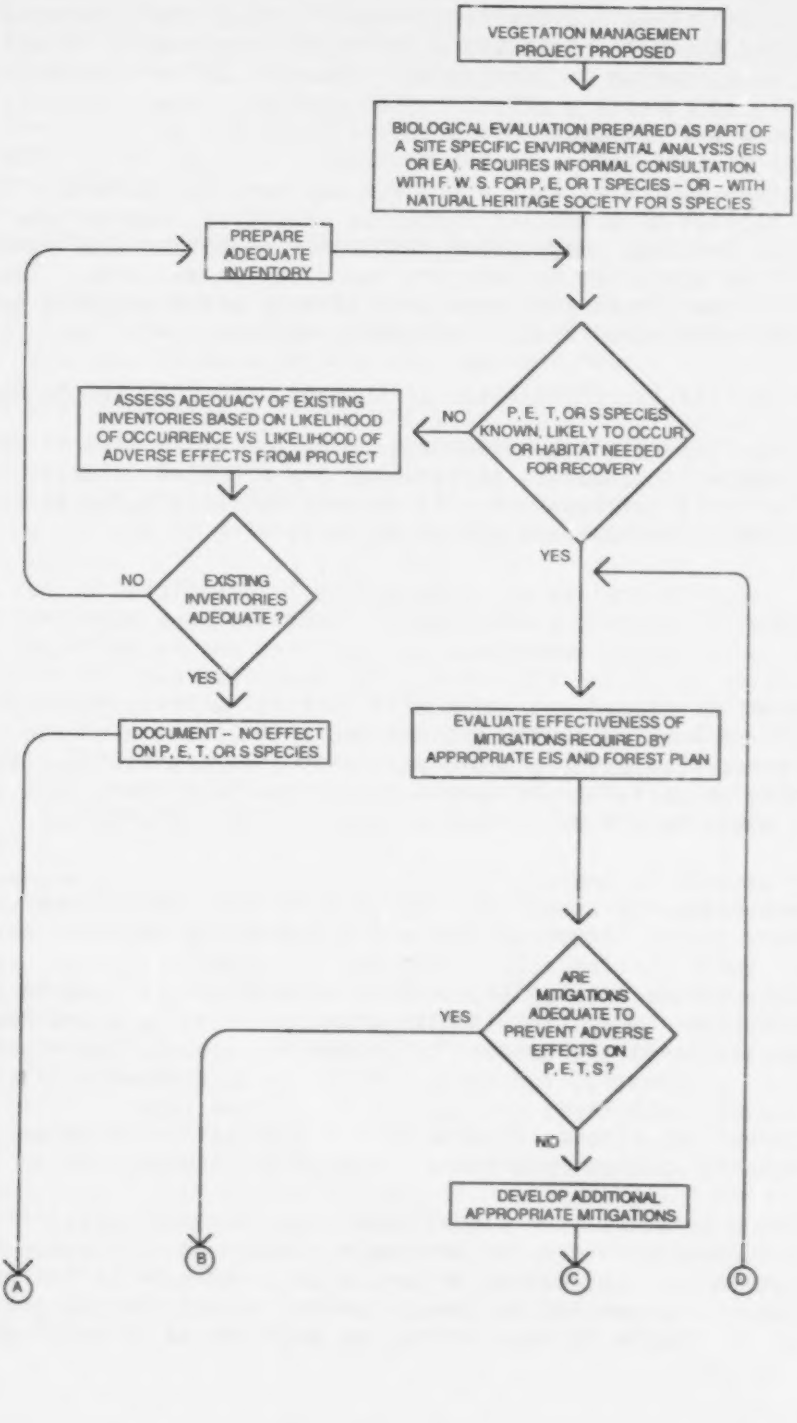
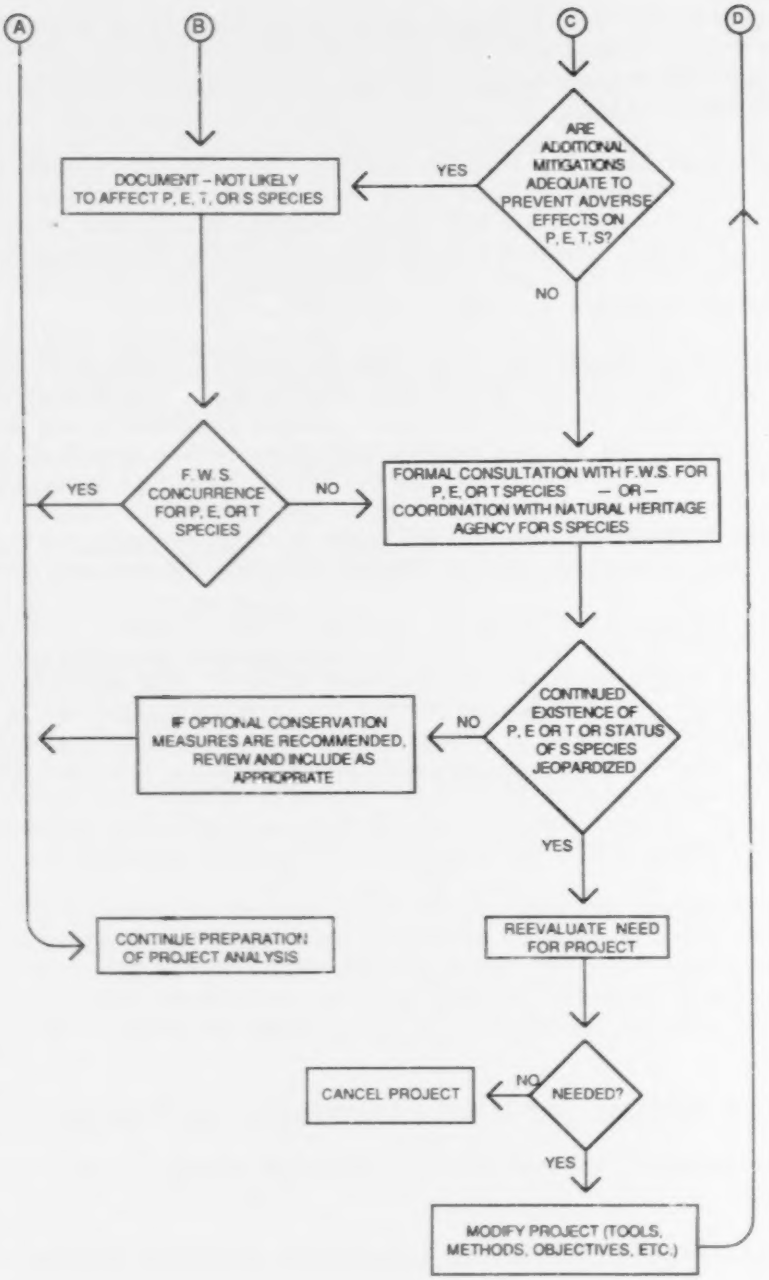

VEGETATION MANAGEMENT PROJECT PROPOSED
BIOLOGICAL EVALUATION PREPARED AS PART OF A SITE SPECIFIC ENVIRONMENTAL ANALYSIS (EIS OR EA). REQUIRES INFORMAL CONSULTATION WITH F. W. S. FOR P, E, OR T SPECIES – OR – WITH NATURAL HERITAGE SOCIETY FOR S SPECIES
PREPARE ADEQUATE INVENTORY
P, E, T, OR S SPECIES KNOWN, LIKELY TO OCCUR OR HABITAT NEEDED FOR RECOVERY
NO
ASSESS ADEQUACY OF EXISTING INVENTORIES BASED ON LIKELIHOOD OF OCCURRENCE VS LIKELIHOOD OF ADVERSE EFFECTS FROM PROJECT
NO
EXISTING INVENTORIES ADEQUATE ?
YES
DOCUMENT – NO EFFECT ON P, E, T, OR S SPECIES
YES
EVALUATE EFFECTIVENESS OF MITIGATIONS REQUIRED BY APPROPRIATE EIS AND FOREST PLAN
YES
ARE MITIGATIONS ADEQUATE TO PREVENT ADVERSE EFFECTS ON P, E, T, S ?
NO
DEVELOP ADDITIONAL APPROPRIATE MITIGATIONS
A
B
C
D

Figure D-1.--Site-specific enviromental analysis process


A
B
C
D
ARE ADDITIONAL MITIGATIONS ADEQUATE TO PREVENT ADVERSE EFFECTS ON P, E, T, S?
YES
DOCUMENT – NOT LIKELY TO AFFECT P, E, T, OR S SPECIES
NO
F. W. S. CONCURRENCE FOR P, E, OR T SPECIES
YES
NO
FORMAL CONSULTATION WITH F.W.S. FOR P, E, OR T SPECIES — OR — COORDINATION WITH NATURAL HERITAGE AGENCY FOR S SPECIES
CONTINUED EXISTENCE OF P, E OR T OR STATUS OF S SPECIES JEOPARDIZED
NO
IF OPTIONAL CONSERVATION MEASURES ARE RECOMMENDED, REVIEW AND INCLUDE AS APPROPRIATE
YES
CONTINUE PREPARATION OF PROJECT ANALYSIS
REEVALUATE NEED FOR PROJECT
NEEDED?
NO
CANCEL PROJECT
YES
MODIFY PROJECT (TOOLS, METHODS, OBJECTIVES, ETC.)

There is a significant risk to the embryos of nesting threatened and endangered birds when kerosene, diesel oil, or any herbicide containing kerosene or diesel oil is broadcast applied during nesting season. A mitigation measure in chapter II prohibits such application.

Table D-2 also shows that one aquatic species, the fat pocketbook pearly mussel, would be adversely affected (significant risk of exposure greater than 1/20 LC_{50}) if an accidental spill released sufficient amounts of certain herbicides into a water body it inhabits. Measures in chapter II of the EIS concerning handling, transportation, application, and spill clean-up mitigate this risk by making the likelihood of such exposure extremely low.

Manual treatments are beneficial when used to improve endangered species habitat. American alligator and the fat pocketbook pearly mussel, are unaffected since treatments do not occur in their habitat. Manual treatments are unlikely to alter aerial insect populations to the extent that gray or Indiana bats would be affected adversely. Other species are protected by Forest Land and Resource Management Plan standards and guides which protect wetlands and streamside zones. The bald eagle and red cockaded woodpecker are not affected as long as nest trees and colony sites are protected, and treatments are performed outside the nesting season.

Soil-disturbing mechanical treatments such as light or heavy disking and bedding may be used to improve the habitat of the red-cockaded woodpecker. They should not be used where the Magazine Mountain shagreen occurs. The American alligator, and fat pocketbook pearly mussel may be harmed if intensive soil-disturbing treatments result in increased siltation of their habitats. Measures in chapter II of the EIS regarding slope restrictions, distance to water bodies, and soil characteristics, reduce siltation and mitigate this risk. Adverse effects on other species are unlikely or do not occur. Non-soil-disturbing mechanical treatments such as mowing and chopping can improve habitat for the red-cockaded woodpecker.

Effects from biological methods generally do not occur since grazing is not conducted in the habitat of most of the listed species. Grazing may be used to improve habitat conditions for red-cockaded woodpecker. Heavy grazing near riparian areas could adversely affect the fat pocketbook pearly mussel. Measures in chapter II of the EIS designed to protect riparian areas from grazing, mitigate these effects.

Potential Adverse Effects--Threatened, Endangered, and Proposed Plants

There are no threatened, endangered, and proposed plants in the Ozark and Ouachita National Forests.

Determination of Effect--Threatened, Endangered, Proposed Species

The actions proposed in the preferred alternative are not likely to adversely affect any threatened, endangered, or proposed species. In accordance with FSM 2670, concurrence with this determination by the USDI Fish and Wildlife Service (FWS) is recommended. The EIS, this biological evaluation, and other appendices will be forwarded to the FWS for their use.

Potential Adverse Effects--Sensitive Animals and Plants

The same general mitigation measures designed to protect threatened, endangered, and proposed species, apply equally to sensitive species except that informal coordination with appropriate State heritage trust agencies takes the place of consultation with the FWS.

To protect Rafinesque's big-eared, small footed, and Eastern Myotis bats, triclopyr is not applied aerially within 300 feet or by ground methods within 60 feet of any known populations.

The eggs of all sensitive bird species are protected from broadcast application of kerosene, diesel oil, and herbicides containing kerosene and diesel oil by restrictions described in chapter II.

All plants listed as sensitive are protected by the same distance restrictions when applying any herbicide.

The effects of other treatments vary from beneficial to no effect to detrimental. These effects are considered in the site-specific environmental assessment and biological evaluation for each project.

Determination of Effect--Sensitive Species

The actions proposed in the preferred alternative are not likely to adversely affect any sensitive species. Informal coordination regarding this determination by appropriate State heritage trust agencies is recommended.

James D. Fenwood

JAMES D. FENWOOD March 1, 1989

Table D-1.--Potential effects of vegetation management (in the absence of mitigating measures) on animal species listed by the U. S. Fish and Wildlife Service as endangered, threatened, or proposed occurring in Ozark and Ouachita National Forests

Common Name	Prescribed Fire	Herbicide	Manual	Soil Disturbing Mechanical	Minimal Soil Disturbing Mechanical	Grazing
Bat, gray	U	U,T	U	U	U	U
Bat, Indiana	U	U,T	U	U	U	U
Eagle, bald	A	A,T	A	A	N	N
Falcon, American peregrine	N	U,T	N	N	N	N
Falcon, Arctic peregrine	N	U,T	N	N	N	N
Woodpecker, red-cockaded	B	A, B,T	B	B	B	B
Alligator, American	N	U,T	U	A	N	A
Mussel, Fat pocketbook pearly	N	U,T	N	A	N	A
Snail, Shagreen, Magazine Mountain	A	T	A	A	A	NA

KEY

A = Adverse habitat effects
B = Beneficial habitat effects if properly applied
T = Significant risk of toxic effects
N = No effect
U = Unlikely
N/A = Not applicable; does not occur

Table D-2.--Chemicals posing potential significant risk (in the absence of mitigation measures) to animal species listed by U. S. Fish and Wildlife Service as endangered, threatened, or proposed occurring in Ozark and Ouachita National Forests. Determination of risk based on risk calculated for most-closely related representative species from risk assessment (shown in parentheses)

Note: Information given within each block applies to all species listed within that block.

Common Name	Broadcast Herbicide (normal rate)	Broadcast Herbicide (extreme rate)	Accident (5-gal. spill)	Accident (100-gal.) spill
Falcon, American peregrine				
Falcon, arctic peregrine	No	TRI	NA	NA
Eagle, bald				
(Kestrel, American)				
Woodpecker, red-cockaded	No	TRI	NA	NA
(Woodpecker, Red-cockaded)				
Bat, gray				
Bat, Indiana	TRI	HEX, TRI	NA	NA
(Bat, red)				
Mussel, fat pocketbook pearly	NA	NA	NA	No
(Oyster, Virginia)				
Alligator, American	No	NA	NA	NA
(E. Box turtle)				

KEY

TRI = Triclopyr
TRI(E) = Triclopyr ester
GLY = Glyphosate
GLY(R) = Glyphosate (Roundup)
HEX = Hexazinone
DIE = Diesel Oil
KER = Kerosene
SUL = Sulfometuron
LIM = Limonene
MIN = Mineral Oil
NO = No Risk
NA = Not Applicable

Table D-3.—Chemicals posing potential significant risk (in the absence of mitigation measures) to animal species classified by Forest Service as sensitive occurring in Ozark and Ouachita National Forests. Determination of risk based on risk calculated for most-closely related representative species from risk assessment (shown in parentheses)

Note: Information given within each block applies to all species listed within that block.

Common Name	Broadcast Herbicide (normal rate)	Broadcast Herbicide (extreme rate)	Accident (5-gal. spill)	Accident (100-gal.) spill
Hawk, Red-shouldered	No	TRI	NA	NA
(Kestrel, American)				
Sparrow, Bachman's	No	NA	NA	NA
Sparrow, rufous-crowned				
(Quail, Bobwhite)				
Salamander, Caddo Mountain	No	TRI	NA	NA
Salamander, four-toed				
Salamander, Fourche Mountain				
Salamander, Oklahoma				
Salamander, Rich Mountain				
Salamander,Southern Red-backed				
(Toad, Woodhouse)				
Turtle, Alligator Snapping	No	NA	NA	NA
(Turtle, Eastern box)				
Mussel, Arkansas fat mucket	NA	NA	DIE, GLY, KER, LIM, TRI(E), SUL	KER, DIE, SUL
Mussel, Neosho mucket				
Mussel, Western fan-shelled pearly				
(Oyster, Virginia)				

Table D-3.—Chemicals posing potential significant risk (in the absence of mitigation measures) to animal species classified by Forest Service as sensitive occurring on Ozark and Ouachita National Forests. Determination of risk based on risk calculated for most-closely related representative species from risk assessment (shown in parentheses) (continued)

Note: Information given within each block applies to all species listed within that block.

Common Name	Broadcast Herbicide (normal rate)	Broadcast Herbicide (extreme rate)	Accident (5-gal. spill)	Accident (100-gal.) spill
Bat, Rafinesque's big-eared	TRI	HEX, TRI	NA	NA
Bat, small-footed				
Bat, Southeastern Myotis				
(Bat, red)				
Shrike, migrant loggerhead	NA	HEX, TEB, TRI	NA	NA
(Bluebird, Eastern)				
Madtom, Caddo Mountain	NA	NA	TRI(E), GLY(R), LIM, SUL	TRI(E), KER, DIE, GLY(R), SUL
Madtom, Ouachita				
(Hogsucker, northern)				
Darter, Arkansas	NA	NA	DIE, KER, SUL, TRI(E)	DIE, KER, SUL, TRI(E)
Darter, crystal				
Darter, longnose				
Darter, poleback				
(Bass, small mouth)				
Shiner, Kiamichi	NA	NA	DIE, GLY, KER, LIM, SUL, TRI(E)	DIE, KER, SUL, TRI(E)
Shiner, Ouachita Mountain				
Shiner, peppered				
(Minnow, flathead)				

Table D-3.--Chemicals posing potential significant risk (in the absence of mitigation measures) to animal species classified by Forest Service as sensitive occurring in Ozark and Ouachita National Forests. Determination of risk based on risk calculated for most-closely related representative species from risk assessment (shown in parentheses) (continued)

Note: Information given within each block applies to all species listed within that block.

Common Name	Broadcast Herbicide (normal rate)	Broadcast Herbicide (extreme rate)	Accident (5-gal. spill)	Accident (100-gal.) spill
Snail, Rich Mountain slitmouth	NA	NA	NA	NA
Beetle, Magazine Mountain mold				
Amphipod, cave				
Amphipod, elevated spring				

KEY

TRI = Triclopyr
TRI(E) = Triclopyr ester
GLY = Glyphosate
GLY(R) = Glyphosate (Roundup)
HEX = Hexazinone
DIE = Diesel Oil
KER = Kerosene
SUL = Sulfometuron
LIM = Limonene
MIN = Mineral Oil
NO = No Risk
NA = Not Applicable

Table D-4.—Potential effects of vegetation management (in the absence of mitigating measures) on animal species classified by the Forest Service as sensitive, and by the U.S. Fish and Wildlife Service as candidate species being considered for possible listing as threatened, endangered or proposed, occurring in the Ouachita and Ozark National Forests

Common Name	Prescribed Fire	Herbicide	Manual	Soil Disturbing Mechanical	Minimal Soil Disturbing Mechanical	Grazing
Amphipod, elevated spring	U	T	U	A	A	N/A
Amphipod, mountain cave	U	T,U	U	A	A	N/A
Bat, Rafinesque's big-eared	A,U	T,U	A	U	U	U
Bat, small-footed	A,U	T,U	A	U	U	U
Bat, Southeastern Myotis	A,U	T,U	A	U	U	U
Beetle, Magazine Mtn. mold	U	T,U	U	U	U	N/A
Darter, Arkansas	N	A,T	U	A	N	A
Darter, crystal	N	A,T	U	A	N	A
Darter, longnose	N	A,T	U	A	N	A
Darter, paleback	A,U	A,T	U	A	N	A
Dragonfly, Ozark snake-tail	A,U	A,T	U	A	A	A
Hawk, red-shouldered	B	N,T	N	N	N	N
Madtom, Caddo	N	A,T	U	A	N	A
Madtom, Ouachita	N	A,T	U	A	N	A
Mussel, Arkansas fat mucket	N	T,U	N	A	N	A
Mussel, Neosho mucket	N	T,U	N	A	N	A
Mussel, Western fan-shelled pearly	N	T,U	N	A	N	A
Paddlefish	N	A,T	N	A	N	A
Salamander, Caddo Mtn	A,U	A,T,U	A,U	A,U	N,U	N/A
Salamander, four-toed	A	A,T,U	A	A	A	N

Table D-4. Potential effects of vegetation management (in the absence of mitigating measures) on animal species classified by the Forest Service as sensitive, and by the U.S. Fish and Wildlife Service as candidate species being considered for possible listing as threatened, endangered or proposed, occurring in the Ouachita and Ozark National Forests (continued)

Common Name	Prescribed Fire	Herbicide	Manual	Soil Disturbing Mechanical	Minimal Soil Disturbing Mechanical	Grazing
Salamander, Fourche Mtn.	A,U	A,T,U	A,U	A,U	N,U	N/A
Salamander, Oklahoma	N	A,T,U	A,U	A,U	N	A
Salamander, Rich Mtn.	A,U	A,T,U	A,U	A,U	N,U	N/A
Salamander, Southern red-backed	A	A,T,U	A	A	A	N
Shiner, Kiamichi	N	A,T	U	A	N	A
Shiner, Ouachita Mtn.	N	A,T	U	A	A	A
Shiner, peppered	N	A,T	U	A	A	A
Shrike, migrant loggerhead	B	B,T	B	B	B	B
Snail, Rich Mtn. slitmouth	A	A,U,T	A,U	A,U	A,U	N/A
Sparrow, Bachman's	B	A,T	B	A	A	N
Sparrow, rufous-crowned	A,U	A,T,U	A,U	A,U	A,U	N/A
Turtle, alligator snapping	N	T,U	U	B	N	B

A = Adverse habitat effects
B = Beneficial habitat effects if properly applied
T = Significant risk of toxic effects
N = No effect
U = Unlikely to occur
N/A = Not applicable; does not occur

Table D-5.—Potential effects of vegetation management (in the absence of mitigating measures) on plant species classified by the Forest Service as sensitive, and by the U.S. Fish and Wildlife Service as candidate species being considered for possible listing as threatened, endangered or proposed, occurring in the Ouachita and Ozark National Forests

SCIENTIFIC NAME	FIRE	MANUAL	SOIL DISTURBING MECHANICAL	MINIMAL SOIL DISTURBING MECHANICAL	GRAZING
Amorpha ouachitensis	B	B	A	A	A
Anemone quinquefolia	B	B	A	A	A
Bartonia paniculata	A	A	A	A	A
Calamogrostis insperata	B	B	A	A	A
Callirhoe papaver bushii	B	B	A	B	A
Cardamine angustata var. *multifida*	A	B	A	A	A
Carex bromoides	A	A	A	A	A
Carex laevivaginata	A	A	A	A	A
Carex latebracteata	A	A	A	A	A
Carex leptalea	A	A	A	A	A
Carex prasina	A	A	A	A	A
Carex pennsylvanica	B	B	A	A	A
Carex stricta	A	A	A	A	A
Carex virescens	A	A	A	A	A
Castanea pumila var. *ozarkensis*	B	B	A	A	N
Cirsium muticum	A	A	A	A	A
Cypripedium kentuckiense	A	A		A	A
Cypripedium reginae	A	A	A	A	A
Delphinium newtonianum	A	B	A	A	A
Delphinium trealeases	A	B	A	A	A
Dennstaedtia punctilobula	A	A	A	A	A

Table D-5.--Potential effects of vegetation management (in the absence of mitigating measures) on plant species classified by the Forest Service as sensitive, and by the U.S. Fish and Wildlife Service as candidate species being considered for possible listing as threatened, endangered or proposed, occurring in the Ouachita and Ozark National Forests (continued)

SCIENTIFIC NAME	FIRE	MANUAL	SOIL DISTURBING MECHANICAL	MINIMAL SOIL DISTURBING MECHANICAL	GRAZING
Dodecatheon frenchii	A	A	A	A	A
Draba aprica	B	B	B	B	B
Dryopteris × *australis*	A	A	A	A	A
Dryopteris celsa	A	A	A	A	A
Dryopteris spinulosa	A	A	A	A	A
Eriocaulon kornickianum	A	B	A	A	A
Erysimum capitatum	B	B	A	A	A
Galium arkansanum var. *publiflorum*	B	B	A	A	A
Gentiana saponaria	A	A	A	A	A
Hedyotis ouachitana	B	B	A	A	A
Heuchera villosa var. *arkansana*	A	A	A	A	A
Hydrastis canadensis	A	A	A	A	A
Isotria verticillata	B	B	A	A	A
Leavenworthia aurea	B	B	A	A	A
Liatris squarrosa var. *compacta*	B	B	A	A	A
Liparis loeselii	A	A	A	A	A
Lycopodium lucidulum	A	A	A	A	A
Mimulus floribundus	A	B	A	A	A
Mitella diphylla	A	A	A	A	A
Neviusia alabamensis	B	B	A	A	A
Osmunda claytoniana	A	A	A	A	A
Parnassia grandifolia	A	A	A	A	A

Table D-5.--Potential effects of vegetation management (in the absence of mitigating measures) on plant species classified by the Forest Service as sensitive, and by the U.S. Fish and Wildlife Service as candidate species being considered for possible listing threatened, endangered or proposed, occurring in the Ouachita and Ozark National Forests (continued)

SCIENTIFIC NAME	FIRE	MANUAL	SOIL DISTURBING MECHANICAL	MINIMAL SOIL DISTURBING MECHANICAL	GRAZING
Paronychia virginia var. *scoparia*	B	B	A	A	A
Penstemon cobaea var. *purpureus*	B	B	A	A	A
Phlox bifida var. *stellaria*	B	B	A	A	A
Polygala polygama	B	B	A	A	A
Quercus shumardii var. *acerifolia*	A	B	A	A	N
Ribes curvatum	A	B	A	A	A
Ribes cynosbati	A	B	A	A	A
Schisandra glabra	A	B	A	A	A
Selaginella arenicola riddellii	B	B	A	A	A
Silene ovata	A	A	A	A	A
Sium suave	A	A	A	A	A
Spiranthes lucida	A	A	A	A	A
Stachys eplingii	A	A	A	A	A
Stenanthium gramineum	A	A	A	A	A
Streptanthus obtusifolius	B	B	A	A	A
Streptanthus squamiformis	B	B	A	A	A
Thelypteris noveboracensis	A	A	A	A	A
Tradescantia ozarkana	A	A	A	A	A
Trichomanes boschianum	A	A	A	A	A
Trichomanes petersii	A	A	A	A	A
Trillium flexipes	A	A	A	A	A

Table D-5.—Potential effects of vegetation management (in the absence of mitigating measures) on plant species classified by the Forest Service as sensitive, and by the U.S. Fish and Wildlife Service as candidate species being considered for possible listing as threatened, endangered or proposed, occurring in the Ouachita and Ozark National Forests (continued)

SCIENTIFIC NAME	FIRE	MANUAL	SOIL DISTURBING MECHANICAL	MINIMAL SOIL DISTURBING MECHANICAL	GRAZING
Trillium pusillum var. *ozarkanum*	A	A	A	A	A
Uvularia perfoliata	A	B	A	A	A
Veratium woodii	B	B	A	A	A
Waldsteinia fragarioides	B	B	A	A	A
Woodsia scopulina var. *appalachiana*	A	A	A	A	A

KEY

A = Adverse habitat effects
B = Beneficial habitat effects if properly applied
N = No effect

Lists of Threatened, Endangered, Proposed, and Sensitive Animal and Plant Species of the Coastal Plain/Piedmont

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APPENDIX E

APPENDIX E

LISTS OF THREATENED, ENDANGERED, PROPOSED, AND SENSITIVE ANIMAL AND PLANT SPECIES OF THE COASTAL PLAIN/PIEDMONT

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Table E-1.--Animal species listed by the U.S. Fish and Wildlife Service as endangered, threatened, or proposed, that occur or may occur in the Ouachita and Ozark National Forests

COMMON NAME	SCIENTIFIC NAME	STATUS	STATES CLASSIFIED T.E.P	HABITAT
Alligator, American	Alligator mississippiensis	T***	AR/OK	Swamps, lakes, marshy areas.
Bat, gray	Myotis grisescens	E	AR	Caves, especially near large streams and nearby forests.
Bat, Indiana	Myotis sodalis	E	AR/OK	Caves and adjoining forests.
Cougar, eastern	Felis concolor couguar	E**	AR	Habitat mosaic of different forest types and successional stages.
Darter, leopard	Percina pantherina	T**	AR	Gravel and cobble-bottomed riffles and raceways of larger creeks and small rivers with high gradients.
Eagle, Bald	Haliaeetus leucocephalus	E	AR/OK	Wintering. Large impoundments, rivers.
Falcon, American peregrine	Falco peregrinus anatum	E	AR/OK	Rare, winter migrant.
Falcon, Arctic peregrine	Falco peregrinus tundruis	T	AR/OK	Rare, winter migrant.
Mussel, Fat pocketbook pearly	Potamilus (=Proptera) capax	E	AR	Large rivers and tributaries. Substrate contains a mixture of sand, mud, and clay.
Panther, Florida	Felis concolor coryi	E**	AR	Habitat mosaic of different forest types and successional stages.
(Snail) Shagreen, Magazine Mountain	Mesodon magazinensis	PT	AR	Cool moist crevices within rock slides on the north slope of Magazine mountain.
Warbler, Bachman's	Vermivora bachmannii	E*		
Woodpecker, ivory-billed	Campephilus principalus	E*		
Woodpecker, red-cockaded	Picoides borealis	E	AR/OK	Open mature stands of shortleaf pine.

*No verifiable sightings on national forest lands in recent history.
**Occurrence questionable. Cooperative study between the Arkansas Game and Fish Commission, USDI Fish and Wildlife Service, and the Ouachita and Ozark National Forests, entitled, "Field Investigation of the Florida Panther In Arkansas Through Radio Telemetry," should resolve the question of occurrence and distribution of the cougar/panther in Arkansas. The leopard darter occurs within the proclaimed boundary of the Ouachita N.F., but has been found only in streams in private ownership. Two intensive studies of upland streams on N.F. have either indicated the habitat unsuitable and/or the fish was not found.
***Threatened due to similarity of appearance.

Table E-2.--Plant species listed by the U.S. Fish and Wildlife Service as endangered, threatened, or proposed, occurring in the Ouachita and Ozark National Forests.

There are no plant species listed as endangered, threatened, or proposed.

Table E-3.--Animal species listed as sensitive by the U.S. Forest Service, and species being considered for possible listing as threatened, endangered, or proposed by the U.S. Fish and Wildlife Service, occurring in the Ouachita and Ozark National Forests

COMMON NAME	SCIENTIFIC NAME	STATES CLASSIFIED CANDIDATE/SENSITIVE	HABITAT
Amphipod, elevated spring	*Stygobromus elatus*	AR	Springs on slope of Magazine Mountain.
Amphipod, mountain cave	*Stygobromus montanus*	AR/OK	Springs on top of Rich Mountain
Bat, Rafinesque's big-eared	*Plecotus rafinesquii*	AR/OK	Caves, forested areas, hollow trees.
Bat, small-footed	*Myotis leibii*	AR/OK	Caves in winter, forested areas.
Bat, Southeastern Myotis	*Myotis austroriparius*	AR	Abandoned mines, caves, hollow trees, forested areas near water.
Beetle, Magazine Mtn. mold	*Arianops sandersoni*	AR	Damp debris at base of bluffs on Magazine Mountain.
Darter, Arkansas	*Etheostoma cragini*	AR	In association with aquatic vegetation in small permanent-flow springs and spring-fed creeks.
Darter, crystal	*Ammocrypta asprella*	OK	Riffle areas in moderate to large rivers, over sandy bottoms.
Darter, longnose	*Percina nasuta*	AR	Clear, silt-free upland streams.
Darter, paleback	*Etheostoma pallididorsum*	AR	Slack-water areas along edges of clear, spring-fed gravel bottomed streams.
Dragonfly, Ozark snake-tail	*Ophiogomphus westfalli*	AR	Riparian areas of small to large streams.

Table E-3.—Animal species listed as sensitive by the U.S. Forest Service, and species being considered for possible listing as threatened, endangered, or proposed by the U.S. Fish and Wildlife Service, occurring in the Ouachita and Ozark National Forests (continued)

COMMON NAME	SCIENTIFIC NAME	STATES CLASSIFIED CANDIDATE/SENSITIVE	HABITAT
Hawk, red-shouldered	Buteo lineatus	AR/OK	Forested areas, particularly mature bottomland forests.
Madtom, Caddo	Noturus taylori	AR	Clear, shallow, water flowing over small rocks or gravel producing shoals near shore.
Madtom, Ouachita	Noturus lachneri	AR	Backwater areas with cobblestone-sized rocks, to small gravel, to soft substrates in clear, high-gradient streams.
Mussel, Arkansas fat mucket	Lampsilis powelli	AR	Clear, silt-free, high-gradient streams.
Mussel, Neosho mucket	Lampsilis rafinesqueana	AR	Clear, silt free, high-gradient streams.
Mussel, Western fan-shelled pearly	Cyprogenia aberti	AR	Clear, silt-free, high-gradient streams.
Paddlefish	Polydon spathula	AR	Large, low-gradient, river systems and their tributaries.
Salamander, Caddo Mtn.	Plethodon caddoensis	AR	Moist hardwood, mixed forests on north facing slopes, under logs and rocks, of the Novaculite Uplift area.
Salamander, four-toed	Hemidactylium scutatum	AR/OK	In moss, under logs and rocks adjacent to springs and seeps.
Salamander, Fourche Mtn.	Plethodon fourchensis	AR	Moist mixed forests on north facing slopes under logs and rocks.
Salamander, Oklahoma	Eurycea tynerensis	AR	Cool, clear creeks and springs with large amounts of gravel for substrate.

Table E-3.—Animal species listed as sensitive by the U.S. Forest Service, and species being considered for possible listing as threatened, endangered, or proposed by the U.S. Fish and Wildlife Service, occurring in the Ouachita and Ozark National Forests (continued)

COMMON NAME	SCIENTIFIC NAME	STATES CLASSIFIED CANDIDATE/SENSITIVE	HABITAT
Salamander, Rich Mtn.	*Plethodon ouachitae*	AR/OK	Moist, hardwood and mixed forests on north slopes under sandstone rubble, logs and occasionally in caves.
Salamander, Southern red-backed	*Plethodon serratus*	AR/OK	Under rocks, logs in riparian areas and on mesic north slopes.
Shiner, Kiamichi	*Notropis ortenburgeri*	AR/OK	Pools over gravel, rubble, or boulder-strewn substrates in small to moderate sized, clear upland streams.
Shiner, Ouachita Mtn.	*Notropis snelsoni*	AR	Pool regions of clear, high-gradient medium to large sized streams.
Shiner, peppered	*Notropis perpallidus*	AR/OK	Pools at the upper end of riffles in larger streams and in head-water areas.
Shrike, migrant loggerhead	*Lanius ludovicianus migrans*	AR/OK	Grass/forb condition, early seral stage habitat conditions.
Snail, Rich Mtn. slitmouth	*Stenotrema pilsbryi*	AR/OK	Rock slides on the north slopes of Blackfork and Rich
Sparrow, Bachman's	*Aimophila aestivalis*	AR/OK	Mature pine and mixed forests with under-story, young pine plantations.
Sparrow, rufous-crowned	*Aimophila ruficeps*	AR	Rocky, cliff-top shrubland on Magazine Mountain.
Turtle, alligator snapping	*Macroclemys temmincki*	AR/OK	Deep sloughs, muddy pools of larger streams and rivers.

Table E-4.--Plant species listed as sensitive by the U.S. Forest Service, and species being considered for possible listing as threatened, endangered, or proposed by the U.S. Fish and Wildlife Service, occurring in the Ouachita and Ozark National Forests

COMMON NAME	SCIENTIFIC NAME	STATES CLASSIFIED CAND/SENSITIVE	HABITAT
Alumroot, Arkansas	*Heuchera villosa* var. *arkansana*	AR	Ledges of calcareous or sandy rock along upland streams and rivers.
Anemone, wood	*Anemone quinquefolia*	AR	Rocky or dry open woods.
Bartonia, twining	*Bartonia paniculata*	AR/OK	Mesic to very wet low woodlands.
Bedstraw	*Galium arkansanum* var. *publiflorum*	AR	Rocky open woodlands, thin soils, novaculite glades.
Bellwort, perfoliate	*Uvularia perfoliata*	AR	Fertile soils on woodland slopes.
Catch-fly, ovate-leaved	*Silene ovata*	AR	Rich, mesic woodlands.
Chinquapin, Ozark	*Castanea pumila* var. *ozarkensis*	AR/OK	Wooded rocky slopes.
Clubmoss, shining	*Lycopodium lucidulum*	AR	Lower talus slopes, narrow ravines, adjacent to streams.
Delphinium, Moore's	*Delphinium newtonianum*	AR	Moist, loamy clay soils soils in shade of upland hardwood forests.
Featherbells, Eastern	*Stenanthium gramineum*	AR	Woodland seeps.
Fern, Allegheny cliff-	*Woodsia scopulina* var. *appalachiana*	AR	Talus, ledges of sandstone outcrops on north slope near summit of Mt. Magazine.
Fern, bristle-	*Trichomanes boschianum*	AR	Under moist, overhanging sandstone outcrops.
Fern, dwarf filmy-	*Trichomanes petersii*	AR	Moist damp ledges in ravines and along streams.

Table E-4.—Plant species listed as sensitive by the U.S. Forest Service, and species being considered for possible listing as threatened, endangered, or proposed by the U.S. Fish and Wildlife Service, occurring in the Ouachita and Ozark National Forests (continued)

COMMON NAME	SCIENTIFIC NAME	STATES CLASSIFIED CAND/SENSITIVE	HABITAT
Fern, hay-scented	*Dennstacdtia punctilobula*	AR	Crevices and ledges of moist, shaded sandstone outcrops.
Fern, interrupted	*Osmunda claytoniana*	AR	Near springs and cave entrances.
Fern, New York	*Thelypteris noveboracensis*	AR	Moist rocky soils of woods and thickets along seeps and streams.
Fern, small log-	*Dryopteris celsa*	AR	Moist to wet shaded areas around springs in rich woodlands.
Fern, small Southern wood	*Dryopteris* × *australis*	AR	Woodland seeps and moist rocky slopes.
Fern, spinulose shield-	*Dryopteris spinulosa*	AR	Moist, wooded slopes.
Gentian, soapwort	*Gentiana saponaria*	AR/OK	Areas of natural seepage, low woods.
Glade Cress, golden	*Leavenworthia aurea*	OK	Rocky glades and barren areas.
Golden seal	*Hydrastis canadensis*	AR	Rich, moist, woodland slopes, ravines, floor of valley woods.
Gooseberry, granite	*Ribes curvatum*	AR	Rocky bluffs and slopes.
Gooseberry, prickly	*Ribes cynosbati*	AR/OK	North facing slopes and woodland ledges.
Grass-of-Parnassus	*Parnassia grandifolia*	AR	Moist ledges along streams, bases of north facing slopes.
Hedge-nettle, stachys	*Stachys eplingii*	AR	Moist to wet low woodlands.
Hellebore, Wood's false	*Veratium woodii*	AR/OK	Mesic north slope hardwood/mixed forests.

Table E-4.—Plant species listed as sensitive by the U.S. Forest Service, and species being considered for possible listing as threatened, endangered, or proposed by the U.S. Fish and Wildlife Service, occurring in the Ouachita and Ozark National Forests (continued)

COMMON NAME	SCIENTIFIC NAME	STATES CLASSIFIED CAND/SENSITIVE	HABITAT
Hedyotis, Ouachita	*Hedyotis ouachitana*	AR	Thin soils, steep bouldery slopes, areas of exposed novaculite.
Indigo, false	*Amorpha ouachitensis*	AR/OK	Riparian glades.
Lady's slipper, showy	*Cypripedium reginae*	AR	Damp areas, wet woodlands, north-facing bluffs.
Lady's slipper, Southern yellow	*Cypripedium kentuckiense*	AR/OK	Mesic north slopes, ravines, seepage areas.
Ladies-tresses, wide-leaved	*Spiranthes lucida*	AR	Base of moist limestone ledges, bluffs bordering creeks.
Larkspur, Trealease's	*Delphinium trealeases*	AR	Limestone glades.
Liatris, scaly gay-feather	*Liatris squarrosa* var. *compacta*	AR	Rocky to sandy soils, dry open glades and woodlands.
Magnolia (Vine), climbing	*Schisandra glabra*	AR	Rich hardwood forests.
Milkwort, purple (racemed)	*Polygala polygama*	OK	Open ground or open woods.
Miterwort	*Mitella diphylla*	AR	North facing wooded limestone or sandstone bluffs, humus covered talus.
Monkeyflower	*Mimulus floribundus*	AR	Damp areas, both open and shaded.
Moss, Riddell's spike-	*Selaginella arenicola riddellii*	AR	Thin soils, exposed novaculite.
Nail-wort, broom	*Paronychia virginia* var. *scoparia*	AR	Dry open woods.
Oak, maple-leaf	*Quercus shumardii* var. *acerifolia*	AR	North slope of Mt. Magazine.
Pagonia, large whorled	*Isotria verticillata*	OK	Dry woodland slopes, ravines.

Table E-4.—Plant species listed as sensitive by the U.S. Forest Service, and species being considered for possible listing as threatened, endangered, or proposed by the U.S. Fish and Wildlife Service, occurring in the Ouachita and Ozark National Forests (continued)

COMMON NAME	SCIENTIFIC NAME	STATES CLASSIFIED CAND/SENSITIVE	HABITAT
Penstemon, purple	Penstemon cobaea var. purpureus	AR	Limestone glades.
Phlox, sand	Phlox bifida var. stellaria	AR	Rocky slopes, sandy soils, bluffs, glades.
Pipewort, small-headed	Eriocaulon kornickianum	AR	Low, moist woodland openings, natural seeps.
Poppy-mallow, Bush's	Callirhoe papaver bushii	AR	Rocky woods, glades, roadsides.
Reed Grass, Ofer Hollow	Calamogrostis insperata	AR	Woodland ravines, open slopes, bluffs.
Sedge	Carex bromoides	AR	Low, wet woodlands, seeps.
Sedge, bristly-stalk	Carex leptalea	AR	Spring branches, base of moist shaded slopes.
Sedge, drooping	Carex prasina	AR	Seeps, pond borders, low, wet woodlands.
Sedge, Pennsylvania	Carex pennsylvanica	AR	Dryer acidic soils of upland wooded slopes.
Sedge, ribbed	Carex virescens	AR	Along springs, streams, wooded north slopes.
Sedge, smooth-sheathed	Carex laevivaginata	AR	Low wet woodlands, near streams, seeps.
Sedge, Waterfall's	Carex latebracteata	AR	Accumulated humus at base of mesic north-facing slopes.
Sedge, upright	Carex stricta	AR	Mesic to low wet woodlands.
Shooting-star, French's	Dodecatheon frenchii	AR	Deep ravines and protected areas.
Snow-wreath, Alabama	Neviusia alabamensis	AR	Woodlands.
Spiderwort, Ozark	Tradescantia ozarkana	AR	Fertile rocky wood-ledges, ravines.

Table E-4.—Plant species listed as sensitive by the U.S. Forest Service, and species being considered for possible listing as threatened, endangered, or proposed by the U.S. Fish and Wildlife Service, occurring in the Ouachita and Ozark National Forests (continued)

COMMON NAME	SCIENTIFIC NAME	STATES CLASSIFIED CAND/SENSITIVE	HABITAT
Strawberry, barren	*Waldsteinia fragarioides*	AR	Moist or dry forested areas.
Thistle, swamp	*Cirsium muticum*	AR	Spring fed, swampy and seepage areas.
Toothwort, bittercrest	*Cardamine angustata* var. *multifida*	AR	Calcareous, mesic woodlands.
Trillium, Ozark least	*Trillium pusillum* var. *ozarkanum*	AR	Humus on woodland ravine slopes, mesic north slopes.
Trillium, white	*Trillium flexipes*	AR	Mesic fertile slopes, woodland seeps.
Twayblade, yellow (Loesel's)	*Liparis loeselii*	AR	Woodland seeps, boggy areas, wet thickets.
Twistflower	*Streptanthus obtusifolius*	AR	Glades, dry sites, road cuts.
Twistflower	*Streptanthus squamiformis*	AR	Rocky hillsides, roadsides.
Water-parsnip, hemlock	*Sium suave*	AR	Wet woodlands, along streams and springs.
Whitlow-grass, open-ground	*Draba aprica*	AR	Woodlands and opening.
Wallflower, Western	*Erysimum capitatum*	AR	Limestone bluffs, glades, rocky ground.